

1 STATE OF MINNESOTA DISTRICT COURT  
2 COUNTY OF RAMSEY SECOND JUDICIAL DISTRICT  
3 - - - - -  
4 The State of Minnesota,  
5 by Hubert H. Humphrey, III,  
6 its attorney general,  
7 and  
8 Blue Cross and Blue Shield  
9 of Minnesota,  
10 Plaintiffs,  
11 vs. File No. C1-94-8565  
12 Philip Morris Incorporated, R.J.  
13 Reynolds Tobacco Company, Brown  
14 & Williamson Tobacco Corporation,  
15 B.A.T. Industries P.L.C., Lorillard  
16 Tobacco Company, The American  
17 Tobacco Company, Liggett Group, Inc.,  
18 The Council for Tobacco Research-U.S.A.,  
19 Inc., and The Tobacco Institute, Inc.,  
20 Defendants.  
21 - - - - -

22 TRANSCRIPT OF PROCEEDINGS  
23 VOLUME 21, PAGES 4024 - 4212  
24 FEBRUARY 17, 1998  
25

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ADVERSE EXAMINATION - THOMAS OSDENE (BY VIDEOTAPE)

1 P R O C E E D I N G S.  
2 THE CLERK: All rise. Ramsey County  
3 District Court is now in session, the Honorable  
4 Kenneth J. Fitzpatrick now presiding.  
5 (Jury enters the courtroom.)  
6 THE CLERK: Please be seated.  
7 THE COURT: Good morning.  
8 (Collective "Good morning.")  
9 MR. CIRESI: Proceed?  
10 Thank you, Your Honor.  
11 (Videotape played.)  
12 MR. CIRESI: Your Honor, the exhibit that  
13 will now be identified in the deposition is trial  
14 Exhibit No. 3681.  
15 (Videotape continued to be played.)  
16 MR. CIRESI: Your Honor, Deposition Exhibit  
17 1504 is Trial Exhibit No. 3683, and we will offer  
18 that.  
19 MR. GARNICK: No objection.  
20 THE COURT: Could you stop the deposition,  
21 please.  
22 (Videotape stopped.)  
23 THE COURT: Trial Exhibit 3683 will be  
24 received.  
25 Could you wait until it's been -- if there's

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1 been an offer, wait for any objection and the  
2 determination by the court, please.  
3 Thank you.

4 (Videotape continued to be played.)  
5 MR. CIRESI: Your Honor, we will offer  
6 Trial Exhibit 3684, which is identified in the  
7 deposition as Exhibit 1505.  
8 MR. GARNICK: No objection.  
9 THE COURT: Court will receive 3684.  
10 (Videotape continued to be played.)  
11 MR. CIRESI: Your Honor, Exhibit 1506 in  
12 the deposition is Trial Exhibit No. 3685, and we  
13 offer it.  
14 MR. GARNICK: No objection.  
15 THE COURT: Court will receive Trial  
16 Exhibit 3685.  
17 (Videotape continued to be played.)  
18 MR. CIRESI: Your Honor, Deposition Exhibit  
19 103 will be offered as Trial Exhibit No. 2513.  
20 MR. GARNICK: No objection.  
21 THE COURT: Court will receive 2513.  
22 (Videotape continued to be played.)  
23 MR. CIRESI: Your Honor, Deposition Exhibit  
24 1514 will be Trial Exhibit No. 3693, and we'd offer  
25 it.

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1 MR. GARNICK: No objection.  
2 THE COURT: Court will receive 3693.  
3 (Videotape continued to be played.)  
4 MR. CIRESI: Your Honor, Deposition Exhibit  
5 126 will be offered as Trial Exhibit No. 2536.  
6 MR. GARNICK: No objection.  
7 THE COURT: Court will receive 2536.  
8 (Videotape continued to be played.)  
9 MR. CIRESI: Your Honor, Deposition Exhibit  
10 1516 will be offered as Trial Exhibit No. 3695.  
11 MR. GARNICK: No objection.  
12 THE COURT: Court will receive 3695.  
13 (Videotape continued to be played.)  
14 MR. CIRESI: Your Honor, the next exhibit  
15 will be Deposition Exhibit 1517, which will be  
16 offered as Trial Exhibit 3696.  
17 MR. GARNICK: No objection.  
18 THE COURT: Court will receive 3696.  
19 (Videotape continued to be played.)  
20 MR. CIRESI: The next exhibit is Deposition  
21 Exhibit 1518, which will be offered as Trial Exhibit  
22 3697.  
23 MR. GARNICK: No objection.  
24 THE COURT: Court will receive 3697.  
25 (Videotape continued to be played.)

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1 MR. GARNICK: Objection to the next  
2 question and answer, Your Honor, as being  
3 inconsistent with the court's order. Counsel is  
4 testifying, and the question relates to periods of  
5 time that go beyond Dr. Osdene's tenure.  
6 THE COURT: The objection is sustained.  
7 MR. CIRESI: The next exhibit, Your Honor,  
8 will be --

9 THE COURT: Counsel, excuse me, counsel.  
10 As I understand it, that's page 93, lines nine  
11 through 12; is that correct?  
12 MR. CIRESI: That is correct.  
13 MR. GARNICK: Yes.  
14 MR. CIRESI: Through 13, Your Honor.  
15 MR. GARNICK: Through 13.  
16 THE COURT: Through 13. I just want the  
17 record to show that.  
18 MR. CIRESI: The next deposition exhibit  
19 will be 1519, which will be offered as Trial Exhibit  
20 3698.  
21 MR. GARNICK: No objection.  
22 THE COURT: Court will receive 3698.  
23 (Videotape continued to be played.)  
24 MR. CIRESI: The next deposition exhibit is  
25 1520, which will be offered as Trial Exhibit 3699.

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1 MR. GARNICK: No objection.  
2 THE COURT: Court will receive 3699.  
3 (Videotape continued to be played.)  
4 MR. GARNICK: Objection. Same basic  
5 grounds as before that is in Your Honor's order, it  
6 goes beyond the document, and also it's not limited  
7 in time to Dr. Osdene's tenure.  
8 MR. CIRESI: Just asking whether he  
9 recalled, Your Honor, which is limited by its  
10 question to the time that's related to the exhibit.  
11 THE COURT: The objection is sustained.  
12 MR. GARNICK: And that would be page 132,  
13 line 20, to page 133, line two.  
14 MR. CIRESI: The next deposition exhibit,  
15 then, would be 145, which will be offered as Trial  
16 Exhibit 2554.  
17 MR. GARNICK: No objection.  
18 THE COURT: Court will receive 2554.  
19 (Tape continued to be played.)  
20 MR. CIRESI: Your Honor, the next  
21 deposition exhibit, 279, is offered as Trial Exhibit  
22 2688.  
23 MR. GARNICK: If they're offering it, we  
24 have no objection.  
25 THE COURT: Court will receive 2688.

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1 (Videotape continued to be played.)  
2 MR. GARNICK: Objection, page 140, line 15  
3 through 21, goes beyond the document, it's not  
4 limited in time to Dr. Osdene's tenure, and it is  
5 testimony of counsel.  
6 MR. CIRESI: It is directly related to the  
7 preceding question, which was the final full  
8 paragraph on the second page, and he's being asked a  
9 question directly related to that paragraph.  
10 THE COURT: You may respond.  
11 (Videotape continued to be played.)  
12 MR. CIRESI: Exhibit 91 is being offered as  
13 Trial Exhibit 2501, Your Honor.

14 MR. GARNICK: No objection.  
15 THE COURT: Court will receive 2501.  
16 (Videotape continued to be played.)  
17 MR. GARNICK: Objection, again goes beyond  
18 the document, beyond that it mischaracterizes Dr.  
19 Charles' testimony. And this objection relates to  
20 page 142, lines 15 through 21.  
21 MR. CIRESI: This is preparatory to Dr.  
22 Charles' testimony which will be offered, Your Honor,  
23 and it's also preparatory to asking him a question  
24 with regard to his recollection, and it relates  
25 specifically to the document in question.

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1 THE COURT: Okay. I'll allow the testimony  
2 as subject to a motion to strike after the testimony  
3 is received of Dr. Charles.

4 (Videotape continued to be played.)  
5 MR. CIRESI: Your Honor, Exhibit 1529 from  
6 the deposition has the Trial Exhibit No. 3708, and  
7 we'll offer Exhibit 3708.

8 MR. GARNICK: No objection.  
9 THE COURT: Court will receive 3708.  
10 (Videotape continued to be played.)  
11 MR. CIRESI: Your Honor, Deposition Exhibit  
12 148 will be offered as Trial Exhibit No. 2557.

13 MR. GARNICK: No objection.  
14 THE COURT: Court will receive 2557.  
15 (Videotape continued to be played.)  
16 THE COURT: We'll be taking a short recess  
17 at this time.

18 THE CLERK: Court stands in recess.  
19 (Videotape paused at deposition page 229,  
20 line 20.)

21 (Recess taken.)  
22 THE CLERK: Ramsey County District Court is  
23 again in session.  
24 (Jury enters the courtroom.)  
25 THE CLERK: Please be seated.

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1 MR. CIRESI: Thank you, Your Honor. We  
2 would offer two previous exhibits that we had not,  
3 Your Honor, but were the subject of Dr. Osdene's  
4 testimony: Exhibit 3680, which was Deposition  
5 Exhibit 1501, and it was at page 37 of the  
6 deposition, and Trial Exhibit 3681, which was  
7 Deposition Exhibit 1502, and it was at page 41 of the  
8 deposition.

9 MR. GARNICK: No objection.  
10 THE COURT: Court will receive 3680 and  
11 3681.

12 MR. CIRESI: And the questions now are by  
13 Philip Morris's attorney, Mr. Webb.

14 (Videotape started at page 230, line 24.)  
15 MR. CIRESI: We have to roll the tape for  
16 the next part, Your Honor; that's why it's taking a  
17 while.

18 (Videotape continued to be played.)

19 MR. CIRESI: That completes the deposition  
20 of Dr. Osdene, Your Honor.

21 We would call Dr. Scott F. Davies to the stand,  
22 Your Honor. Dr. Davies.

23 (Witness sworn.)

24 THE CLERK: Please state your name and  
25 spell your last name for the record.

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1 THE WITNESS: Scott F. Davies, D-a-v-i-e-s.

2 MR. CIRESI: Doctor, you may want to attach  
3 that to your belt. The other -- the other part,  
4 not -- there you go.

5 SCOTT F. DAVIES

6 called as a witness, being first duly  
7 sworn, was examined and testified as  
8 follows:

9 DIRECT EXAMINATION

10 BY MR. CIRESI:

11 Q. Good morning, doctor.

12 A. Good morning.

13 Q. Doctor, you reside at DELETED

14

15 A. Yes, I do.

16 Q. And you're presently the director, Division of  
17 Pulmonary and Critical Care Medicine, Department of  
18 Internal Medicine at the Hennepin County Medical  
19 Center in Minneapolis?

20 A. Yes, I am.

21 Q. And you're also the medical director, chief of  
22 medical staff at Vencor Hospital in Golden Valley,  
23 Minnesota?

24 A. Yes, I am.

25 Q. Doctor, you're here to testify about chronic

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1 obstructive pulmonary disease, one of the major  
2 smoking-caused diseases?

3 A. Yes, I am.

4 Q. Doctor, before we get to your testimony itself,  
5 I'd like you to review just briefly your background  
6 and education for the jury and the court.

7 You obtained your B.A. degree from the College  
8 of Holy Cross in Massachusetts?

9 A. Yes, I did.

10 Q. And then you obtained your M.D. in 1974 from the  
11 University of Minnesota?

12 A. Yes.

13 Q. And from 1974 to 1975, you were an intern in  
14 internal medicine at the University of Minnesota?

15 A. That's correct.

16 Q. And from 1975 to 1977, you were a resident in  
17 internal medicine at the University of Minnesota;  
18 correct?

19 A. Correct.

20 Q. With regard to your licensing and  
21 certifications, you're a diplomat of the American  
22 Board of Internal Medicine?

23 A. Yes, I am.

24 Q. You're also a member of the Pulmonary Medicine  
25 Subspecialty Boards?

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1 A. Yes.

2 Q. And you have critical care certification;  
3 correct?

4 A. Correct.

5 Q. And from 1979 to 1994 you were an assistant  
6 professor of medicine at the University of Minnesota  
7 Medical School?

8 A. Yes.

9 Q. And 1985 to 1994 you were an associate professor  
10 of medicine at the University of Minnesota Medical  
11 School?

12 A. Yes.

13 Q. And from 1994 to the present time you are a  
14 professor of medicine at the university's medical  
15 school; correct?

16 A. Yes.

17 Q. And at the present time you are a course  
18 director at the University of Minnesota Medical  
19 School in a course entitled "Pathophysiology,  
20 Respiratory Medicine?"

21 A. Yes, I am.

22 Q. And 1979 to the present time you've also been on  
23 the faculty for clinical rotations in pulmonary  
24 medicine and critical care medicine at the Hennepin  
25 County Medical Center for medical students, medical

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1 residents and pulmonary fellows and critical care  
2 fellows; is that correct?

3 A. That is correct.

4 Q. Can you describe that program a little bit,  
5 please, doctor.

6 A. Well Hennepin County Medical Center is a  
7 teaching hospital, one of the four major teaching  
8 hospitals of the University of Minnesota. It's a  
9 urban, city hospital, and as medical students in  
10 their last two years of their training get clinical  
11 experience, they rotate and take different rotations  
12 or elective rotations at our hospital. And for  
13 example, they might do six weeks on an internal  
14 medicine ward, and they might do six weeks in an  
15 intensive care unit, and they might do six weeks  
16 studying pulmonary diseases and that -- by seeing  
17 patients and working with the faculty.

18 The patients in the hospital are generally taken  
19 care of by a team, and that team has often a medical  
20 student, and then a resident, who's someone who has  
21 finished medical school but training to become  
22 boarded in internal medicine, and then a faculty  
23 member, working together to take care of that  
24 patient. And so the students -- about 35 percent of  
25 all clinical rotations of Minnesota -- University of

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- 1 Minnesota students are spent at our hospital. It's  
2 one of the major teaching sites. And I work with  
3 those students and those residents, but in the  
4 process of taking care of the patients who come for  
5 various different problems.
- 6 Q. And doctor, from 1980 up to the present time,  
7 how many students at the University of Minnesota  
8 Medical School have you taught respiratory medicine  
9 to?
- 10 A. The second-year course that Mr. Ciresi is  
11 referring to is a lecture-type course that goes six  
12 weeks in the second -- in the fall of the second year  
13 of the medical school, and all the students take that  
14 course; it's their introduction to lung diseases.  
15 And there's about 200 students, and I've taught the  
16 course for 18 years, so basically every student who's  
17 come through, over 3,000 students would have taken  
18 that course since 1980.
- 19 Q. And do you also serve on the faculty at many  
20 local and regional post-graduate courses sponsored by  
21 various institutions?
- 22 A. Yes, I have.
- 23 Q. And those institutions would include the Mayo  
24 Clinic?
- 25 A. Yes.

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- 1 Q. The University of Michigan?
- 2 A. Yes.
- 3 Q. And the University of North Dakota?
- 4 A. Yes.
- 5 Q. And doctor, are you also a member in various  
6 professional societies?
- 7 A. I am.
- 8 Q. Does that include the American Thoracic Society?
- 9 A. Yes, it does.
- 10 Q. And the American College of Chest Physicians?
- 11 A. Yes.
- 12 Q. And have you also held various offices and  
13 committee assignments in the professional societies  
14 that you belong to?
- 15 A. Yes, I have.
- 16 Q. Have you been the chairman of the  
17 Cardiopulmonary Infection Steering Committee of the  
18 American College of Chest Physicians?
- 19 A. Yes, I have.
- 20 Q. Have you also been the president of the  
21 Minnesota Thoracic Society?
- 22 A. Yes, I have.
- 23 Q. Have you also been on the Tuberculosis and  
24 Pulmonary Infection Program Committee of the American  
25 Thoracic Society, Scientific Assembly on

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- 1 Microbiology?
- 2 A. Yes, I have.
- 3 Q. And in 1987 to 1982 did you serve as the  
4 Governor for the State of Minnesota, the American

5 College of Chest Physicians?

6 A. Yes, I did.

7 Q. And doctor, with respect to your primary areas  
8 of research at the present time, does that include  
9 deep fungal infections?

10 A. Yes, it does.

11 Q. And can you describe what that is, please.

12 A. There are a variety of chronic pneumonias. Most  
13 pneumonias are -- we think of, you take an antibiotic  
14 and the pneumonia goes away in a week or two, and  
15 there's some chronic infections in the lung that last  
16 a lot longer and require different antibiotics over a  
17 longer period of time, and the one that's most common  
18 would be tuberculosis that most people would know  
19 about that doesn't go away in a week like a  
20 pneumonia, it requires many drugs for many months,  
21 and it's sort of more subacute or chronic. There are  
22 other chronic pneumonias caused by fungal organisms  
23 that you inhale from the soil. Some of them are  
24 common in southern Minnesota and northern Minnesota.  
25 And they also present like -- not like a bacterial

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1 pneumonia where it goes away in a week with an  
2 antibiotic, but requiring many months of treatment  
3 and presenting a little bit differently. And I've  
4 studied the epidemiology of those diseases and the  
5 treatment and various clinical -- clinically-related  
6 aspects of those diseases.

7 Q. And have you also done major research in  
8 obstructive sleep apnea?

9 A. I'm part of the faculty at the Minnesota  
10 Regional Sleep Disorder Center, which has been in  
11 existence since 1978, and it's done a lot of research  
12 learning different things about sleep disorders.

13 Q. And you can tell us what sleep apnea, doctor?

14 A. Sleep apnea is sort of -- everyone has seen  
15 someone snore. It's the far end of snoring where the  
16 snoring gets so loud that it's not just rattling the  
17 room, but the patient actually sort of chokes and  
18 obstructs and then has to wake themselves up again to  
19 get breathing again. And those snorers get very  
20 tired during day because their sleep is so fragmented  
21 and choppy and disruptive. That's what sleep apnea  
22 is.

23 Q. And doctor, during the course of your career  
24 have you published in excess of a hundred articles in  
25 peer-reviewed journals and books that are used by

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1 other physicians?

2 A. Yes, I have.

3 Q. And with respect to your practice itself,  
4 doctor, is about 20 percent of it spent in treating  
5 chronic obstructive pulmonary disease?

6 A. Ten to 20 percent of my inpatient practice and a  
7 slightly higher percentage of my outpatient practice  
8 would be seeing patients with chronic obstructive  
9 lung disease. I see them every single day when I'm

10 in the hospital and every single day when I'm in the  
11 clinic, patients with this problem.  
12 Q. Doctor, can you describe what is chronic  
13 obstructive pulmonary disease?  
14 A. The -- the pulmonary part, "pulmonary" just  
15 means lung, so it's a lung disease, and the "disease"  
16 itself is explanatory. What "obstructive" means is  
17 that there's a problem with emptying the lungs. The  
18 lungs can't empty normally, so there's obstruction to  
19 air flow during exhaling, exhaling is impeded and  
20 slowed. And the "chronic" means that the condition  
21 is permanent, that it cannot go away. It can partly  
22 get better sometimes, but it cannot go away. So  
23 "chronic" means a permanent condition in which  
24 there's obstruction or slowing of expiration, the  
25 patients can't exhale normally. And the "pulmonary"

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1 is just that it's the -- it involves the lung and  
2 that it is a major disease.  
3 Q. Doctor, how does that compare with like asthma?  
4 A. Asthma also has airflow obstruction and patients  
5 with asthma can't empty their lungs. And it's a  
6 pulmonary disease. But asthma, the obstruction is  
7 due to spasm of the smooth muscles around the little  
8 air tubes, and secretions, so that it is not a  
9 chronic disease, it can -- in the sense that an  
10 attack of asthma can reverse. And someone with  
11 asthma can come in at another time when they've been  
12 well treated and are not having symptoms and have  
13 normal lung function with no obstruction. So it's a  
14 reversible obstructive airway disease would be  
15 asthma, whereas COPD is a chronic, irreversible sort  
16 of like an asthma attack that can never end, that  
17 will last forever.  
18 Q. Doctor, we've had Drs. Hurt and Dr. Samet and  
19 Dr. Robertson describe for the jury and the court the  
20 anatomy of the lung. But I'd like to have you touch  
21 briefly on it with respect to how it will impact your  
22 testimony here.

23 Could you go to the exhibit book in front of you  
24 and please direct your attention to Exhibit 30054.

25 MR. CIRESI: Which we would offer for  
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1 illustrative purposes, Your Honor.  
2 MR. MONICA: Your Honor, I object to  
3 counsel summarizing or attempting to summarize  
4 testimony from prior witnesses and ask that counsel  
5 be instructed to just ask direct questions.  
6 THE COURT: Well I think the question is  
7 pretty preliminary.  
8 MR. CIRESI: It was, Your Honor.  
9 THE COURT: I'll let it stand.  
10 MR. CIRESI: Is there an objection to 30054  
11 for illustrative purposes?  
12 MR. MONICA: There is no objection.  
13 THE COURT: The court will receive 30054.  
14 BY MR. CIRESI:

15 Q. Doctor, if we can start with maybe the depiction  
16 in the upper third and work our way down, and if you  
17 could describe what is being represented by this  
18 exhibit.

19 A. It's my understanding that you've been shown  
20 some normal anatomy of the lungs and how the windpipe  
21 is the biggest tube, and then it divides into  
22 ever-smaller tubes, so there's a series of branching  
23 tubes that carry the air out into the lung tissue.  
24 And where we're starting, up on the right part --  
25 this won't show on the screen, but if we start on the

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1 very upper right is one of the very small tubes  
2 that's already branched 15 times, so it's getting  
3 through the -- what's called the conducting airways  
4 or the tubes that bring the airway to the lung and  
5 it's getting close to the business end of the lung  
6 where the oxygen actually goes into the blood and the  
7 carbon dioxide comes out into the gas so you can  
8 exhale it. This last -- the bronchus at the upper  
9 right entering the screen is labeled "Terminal  
10 Bronchiole," and that's because it's one of the small  
11 terminal bronchioles that leads to the business end of  
12 the lung where the gas exchange comes to. And then  
13 you can see that the next branch of tubes has a few  
14 little sacs off the edges, it starts to get little  
15 tiny sacs around the edges, not all the way along,  
16 but sort of scattered along that tube, and that's  
17 called the respirator bronchiole, of which there are  
18 several branchings of that. So it means they have  
19 some parts in gas exchange because there's little  
20 sacs, air sacs actually coming off the tubes, and  
21 then it goes all the way to the end, you can see that  
22 the little air spaces have sacs all the way around  
23 them, and that's all they do. It's done conducting  
24 air down, and that's just where the air sits and gas  
25 exchange occurs. So this is sort of the terminal,

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1 business-end of the lung.

2 Q. And doctor, at the end you were talking about  
3 the alveolar sacs?

4 A. The alveolar sacs are these little tiny sort of  
5 blisters around the edge of those round alveolar  
6 ducts at the end.

7 Q. Can you then describe what is being depicted in  
8 the middle third, doctor, of this Exhibit 30054.

9 A. What --

10 Can we come down just one second to the top  
11 again?

12 Q. Yes.

13 A. Because what -- what's important is that when we  
14 talk about obstruction, or patients not being able to  
15 exhale the air from their lungs, it's chronic  
16 obstructive lung disease, and where that happens is  
17 in these very small airways, the very small airways  
18 are -- that sort of limiting factor for air getting  
19 out of this lung and back up into the windpipe and

20 the other bigger pipes that carry it out of -- out of  
21 the lung. And the -- the -- so that right where you  
22 see the respiratory bronchioles is where the  
23 obstruction and the problem with can't exhale  
24 develops. Patients can't exhale because of problems  
25 right in that particular area.

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1 And the two kinds of problems that occur are  
2 both related to smoking, and the first is that there  
3 are inflammatory cells that gather in that area,  
4 inflammatory cells just like would gather in a boil,  
5 if you had a boil on your skin they gather there, and  
6 they can cause scarring and secretions and narrowing,  
7 and that can sort of block the exit of air from the  
8 lungs. And the second thing that happens is the lung  
9 starts to dissolve right at that point, and the --

10 In fact, expansion of these air spaces beyond  
11 the terminal bronchiole is what the definition of  
12 emphysema is, and you've all heard the word  
13 "emphysema." It's just large air spaces beyond the  
14 terminal bronchioles.

15 So that if we can go up to the second picture,  
16 this shows what's called centrilobular emphysema,  
17 which is just called that because it's right where  
18 these -- these small air tubes come off the  
19 respiratory bronchiole, before you get out to the  
20 little alveolar sacs at the end. And as the lung  
21 tissue gets dissolved, these spaces get larger and  
22 larger and larger. So that -- normally they're  
23 microscopic, and they become so large that they're  
24 even visible to the naked eye as the lung expands.

25 The next -- the bottom one just shows what's

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1 called panlobular emphysema, where the whole air  
2 space is totally dissolved, not only the proximal  
3 tubes, but all the way out, involving the little tiny  
4 air sacs. And it's changes in this -- in this lung,  
5 it's -- it's anatomic and pathological changes in  
6 this lung that make it not empty, as I'll show you in  
7 a minute. But this is where the action occurs, and  
8 this is why patients have obstructions. Can't --

9 Chronic obstructive lung disease, the  
10 obstruction is cannot empty the lung of air, and the  
11 chronic means it's an irreversible condition.

12 Q. Doctor, can you direct your attention now to  
13 Exhibit 30056.

14 MR. CIRESI: Which again, Your Honor, we'd  
15 offer for illustrative purposes.

16 MR. MONICA: No objection.

17 THE COURT: Court will receive 30056 for  
18 illustrative purposes.

19 A. Now there is a little schematic that's just  
20 taking one of those respiratory bronchioles that were  
21 coming off -- the first branch that were coming off  
22 there, starting to get little air sacs, entering  
23 the -- the business unit of the lung, and cutting it  
24 in side -- sideways, so it's a cross-section of one

25 of these little terminal bronchioles. It's a  
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1 schematic, I drew it, and it's not an actual picture  
2 of tissue, but it illustrates what's happening at  
3 that point in the lung.

4 So it says at the top COPD is an obstructive  
5 lung disease, that means the lungs cannot empty and  
6 that the obstruction, the site of major obstruction  
7 occurs in these small peripheral airways. And it has  
8 several features why this lung won't empty. Normally  
9 when you just relax your lungs -- lungs empty all by  
10 themselves. One of the reasons is that there's all  
11 these inflammatory cells that gather in and around  
12 this airway, and they're marked by little white  
13 circles, and they cause swelling and they cause edema  
14 and they cause scarring.

15 Q. What's edema, doctor?

16 A. Edema is just swelling in the tissue like you  
17 might get if you get a burn, and right over a  
18 welt -- you know, fluid would come into the tissue  
19 and you can see it and it would be raised over your  
20 skin.

21 And the infection-fighting cells are exactly  
22 what would happen if you have a boil, and the boil,  
23 you know, if you puncture it, there's yellow pus in  
24 there, and the cells come in and sit around the  
25 airway like that, and even through the wall of the

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1 airway. And then in the center I'm trying to show  
2 that there are secretions and pus that are in the  
3 airway. And you can hear people cough this up when  
4 they have -- when they have to get that deep phlegm  
5 out of these airways.

6 So that the two things that this inflammation at  
7 this site does is it causes fibrosis and scarring on  
8 this airway, which narrows it, and that keeps the  
9 lung from emptying. But it also -- these cells are  
10 loaded with elastases, and elastases are like meat  
11 tenderizer and they break down various tissue  
12 structures. And what they will do is start  
13 dissolving some of these alveolar walls that normally  
14 connect to these small airways. Because the airways  
15 have to go through the lung, and so there's lung  
16 tissue around them, and normally that lung tissue  
17 helps hold them open when you breathe out. And as  
18 these alveolar walls are destroyed by the elastases,  
19 that's how you get these bigger spaces in the same  
20 area, these air spaces that are emphysema.

21 So when that happens, when these airways are no  
22 longer tethered open by tissue, then they tend to be  
23 floppy and collapse as you breathe out. So that's  
24 the second way in which obstruction occurs, is that  
25 these small airways are not supported, they're not

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1 tethered open, so they tend to narrow and collapse as  
2 the patient breathes out. That's why it's an  
3 obstructive disease.

4 Q. Doctor, we've all had the experience of blowing  
5 up a balloon and it's elastic.

6 A. Right.

7 Q. And then it loses its -- its resilience, if you  
8 will.

9 A. Right.

10 Q. Is there any analogous situation?

11 A. I think on the next slide I was going to show  
12 exactly that.

13 Q. Let me direct your attention, then, to Exhibit  
14 30053.

15 MR. CIRESI: And we'd offer that for  
16 illustrative purposes, Your Honor.

17 MR. MONICA: No objection.

18 THE COURT: Court will receive 30053 for  
19 illustrative purposes.

20 A. So in the --

21 On the top is just the normal situation, again  
22 very schematic. Here there's only two alveoli or two  
23 air spaces being shown, when in reality there's a  
24 hundred million of them. So it's a two-compartment  
25 little schematic. And it shows that that's what the

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1 normal lung looks like, and the pipes leading to this  
2 air space are fairly rigid and they're not narrowed,  
3 and they're also stretched open by the tethering  
4 tissue around them. Also, the alveoli themselves, as  
5 they're stretched, have an elastic property like a  
6 balloon, so that they tend to want to empty as soon  
7 as you've inflated your lungs. You do the work  
8 taking the breath in, and you just relax and the  
9 breath empties very naturally without you feeling it  
10 or doing any work or using any muscles, and part of  
11 that is you stretch the lung and it's like a balloon,  
12 and then the pipes are open, and the pipes are well  
13 supported and the pipes aren't narrow, and the air  
14 just comes out very naturally without any sense of  
15 work.

16 Now the bottom two slides show what happens in  
17 COPD. And on the left is illustrated simply the fact  
18 that if all this inflammation of these tiny airways  
19 causes scarring and secretions and narrowing, that  
20 those balloons are not going to be able to empty  
21 through those narrowed pipes. So it's a disease  
22 where you cannot exhale. And on the right shows you  
23 an area of the lung where the supporting tissue has  
24 been dissolved, the alveolar walls have been  
25 dissolved, and these air spaces are -- are bigger and

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1 larger.

2 And that has two different effects. One is that  
3 the airways aren't tethered open by tissues so they  
4 tend to just collapse and are floppy, but the other  
5 effect is that the balloon has lost all of its

6 elastic properties, so it can't empty partly because  
7 it's just distended, and so all of the walls and all  
8 the elastic tissue has been dissolved, and just  
9 like -- thinking of a rubber balloon that's made of  
10 thick rubber and is very hard to blow up, it's going  
11 to empty very easily, whereas if you made a balloon  
12 of something that was like -- more like Saran Wrap  
13 and didn't have any elastic, it's just going to sit  
14 there and it's not going to empty very well.

15 So there's three main factors related to the  
16 can't empty part, and all of them are permanent,  
17 that's why we call it chronic obstructive lung  
18 disease, and the obstruction is related to three  
19 factors, scarring and secretions and narrowing of  
20 these small airways due to the inflammatory cells,  
21 lots of tethering due to the dissolving of the little  
22 connections that hold the airways open, due again to  
23 the inflammatory cells that gather there, and finally  
24 destruction of the lung so it becomes a balloon that  
25 hasn't got thick rubber any more and it can't sort of

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1 empty forcefully when you relax.

2 And for all those reasons, what we're used to is  
3 taking a breath again, and with an active use of our  
4 diaphragm and our muscles, and then relaxing and the  
5 lung will just empty, nice and normal. And in these  
6 patients, they can not empty, the lungs can't empty  
7 normally, and that's what the word "obstruction" is  
8 talking about.

9 Q. Now doctor, do we have some human lungs here  
10 that were freeze dried to show a normal lung and one  
11 with emphysema?

12 A. Yes, we did.

13 Q. Could you step down, please.

14 MR. CIRESI: Your Honor, could the doctor  
15 step down?

16 Q. Maybe you could -- I could move this up for you,  
17 doctor.

18 A. Well these are --

19 It's interesting, I saw some of the videos you  
20 showed and they looked like just this. But these are  
21 real --

22 MR. MONICA: Your Honor, excuse me, may I  
23 lodge an objection, please? Your Honor, these  
24 lungs, a foundation has not been established as to  
25 where they came from and whose lungs they are, and I

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1 don't know that this witness has that knowledge, and  
2 I would ask that before they be shown to the jury and  
3 discussed, that that be established.

4 MR. CIRESI: Your Honor, these are only  
5 being used for illustrative experience. The doctor  
6 has had experience in dealing with lungs his entire  
7 career, and they're being used for illustrative  
8 purposes only to explain to the jury the concept that  
9 he's just shown on the illustrative depictions that  
10 we've put in, Exhibits 30054, 30056 and 30053.

11 THE COURT: I think it's appropriate so  
12 long as it's just for illustrative purposes.  
13 MR. MONICA: Your Honor, --  
14 MR. CIRESI: Your Honor.  
15 MR. MONICA: -- excuse me, may I go over?  
16 THE COURT: Please.  
17 MR. CIRESI: The normal lung then is  
18 Exhibit 30269, 30269, and it's being offered for  
19 illustrative purposes only. And he also has a sliced  
20 part of the lung, which is Exhibit 30270.  
21 Go ahead, doctor.  
22 A. Okay. First piece is the lung, and it looks  
23 like sponge, and you can actually feel it. It's  
24 not -- it's sterile and it's not going to cause any  
25 harm.

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1 Because these little air sacs are very  
2 microscopic, they're tiny, tiny air sacs, just like  
3 in a sponge has microscopic, and a sponge has a lot  
4 of sponge to it, a lot of tissue to it, and the same  
5 is with these lungs, so they have a fairly good  
6 structure. And basically as you breathe in through  
7 the little air tubes, all the little air sacs  
8 inflate, and then you relax and everything empties  
9 out to its resting position just naturally with no  
10 trouble.  
11 Q. Doctor, could you just maybe move around this  
12 side --  
13 A. Sure.  
14 Q. -- so the court and all the jury can see it.  
15 A. Now this is a piece of a similar lung, a normal  
16 lung, that's just been sliced with a saw, and what  
17 you can do in looking at this is that you can't even  
18 see the air sacs. They're so tiny that they're  
19 really microscopic, the things I've been showing you  
20 on the screen, they're invisible, just like a sponge,  
21 you can't look at it and see the individual holes in  
22 the sponge, even though you know when you squeeze it  
23 and open it, it's going to hold water in those little  
24 holes. You can see some of the --  
25 You will see there are holes here in the center,

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1 and these are the blood vessels that carry blood to  
2 the lung or they're the bigger air tubes that branch  
3 through the lung, getting out to the small -- small  
4 part. And the units that I'm showing you  
5 schematically are very tiny little terminal units of  
6 which there are about 60,000 units supplied by one of  
7 those, what I showed, terminal bronchioles up in the  
8 right upper corner of that. And you can all look at  
9 this and -- and see that there really is --  
10 Maybe I could pass it around in the tray.  
11 Q. If you put it in the box. If we could, Your  
12 Honor.  
13 A. And you can look at this and see that there  
14 really is not any visible holes, that the holes are  
15 sort of beyond --

16 Q. Doctor, the clerk will --  
17 You have to let the clerk do that.  
18 (Clerk displays the exhibit to the jury.)  
19 A. I noticed that none of you wanted to really  
20 touch it, but if you push on this with your thumb,  
21 and it's sort of firm, it's like a firm sponge, and  
22 it has tissue to it.  
23 Q. And doctor, do you also have a freeze dried lung  
24 of emphysema?  
25 A. We have another lung prepared in the same manner  
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1 of a patient who died with severe emphysema from --  
2 who was a long smoker.  
3 MR. CIRESI: Your Honor, for illustrative  
4 purposes, the doctor will be referring to the  
5 Exhibits 30272 and 30273.  
6 A. Now this --  
7 MR. MONICA: Your -- Your Honor, excuse me,  
8 we object to these two exhibits. The witness has  
9 stated one of them, at least, is from a smoker. That  
10 has not been established. I think a foundation needs  
11 to be established. I'm not even sure whether this  
12 witness has ever seen these before today. And we  
13 object to them on that basis.  
14 THE COURT: I think you should establish  
15 some foundation.  
16 BY MR. CIRESI:  
17 Q. Doctor, how do you know that that's a smoker's  
18 lung?  
19 A. These are from the collection of the pathology  
20 laboratory at the University of Minnesota, and the  
21 person who has custody of this collection told me  
22 that he had in his record that this patient was a  
23 smoker.  
24 MR. MONICA: Your Honor, I --  
25 MR. CIRESI: We offer it for illustrative  
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1 purposes, Your Honor.  
2 MR. MONICA: Your Honor, I object. It's  
3 based upon hearsay, and the witness has no first-hand  
4 knowledge of these particular lungs.  
5 THE COURT: Okay. I'll allow it for  
6 illustrative purposes only.  
7 MR. CIRESI: Doctor, make sure you turn  
8 around so everybody can see.  
9 A. Yes, I'm sorry. So this is a lung fixed in the  
10 exact identical manner, and this is the whole lung,  
11 which doesn't illustrate too much, except that a lot  
12 of the tissue has been dissolved, and you can see  
13 that it's crinkly and does not have very much  
14 substance to it, and that's because these small  
15 alveolar sacs have been dissolved and that the spaces  
16 with air in it are much, much bigger, and they aren't  
17 these microscopic. And that's what emphysema does,  
18 is it dissolves the lung tissue. Now we couldn't --  
19 We prepared some sections here, and they're in  
20 plastic bags, and you'll notice that the others

21 aren't in plastic bags, and that's because they're so  
22 much more fragile that they would fall apart if they  
23 weren't in a plastic bag. And when you see at these  
24 sections you can actually see the visible holes in  
25 the terminal air spaces in the lung that you couldn't

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1 see before. When I pass them around, you can just  
2 glance at the outer rim and see that there are holes  
3 that are so big that they're totally visible to the  
4 naked eye. And if you hold it up to the light, you  
5 can actually see through it; it's a moth-eaten,  
6 dissolved lung where all the tissue that's supposed  
7 to form these alveolar walls and hold the gas  
8 exchange units in the lung has been dissolved. And  
9 this is what emphysema is, it is a dissolving of all  
10 these spaces so that the lung becomes moth-eaten and  
11 the spaces -- the air spaces beyond the terminal  
12 bronchiole are enlarged many times beyond what these  
13 normal spaces would be enlarged. And that's what  
14 contributes in the three ways that I told you about  
15 the inability of this lung to empty. The lung cannot  
16 empty. It's an obstructive disease where the  
17 patients cannot exhale any more and cannot release  
18 the lung -- the air from their lungs because the  
19 small air tubes have been narrowed and because  
20 there's bigger air spaces so that the -- what air  
21 tubes there are are not tethered open by the  
22 structure and the tissue of the lung, and because  
23 there's no elastic recoil. The other lung snaps back  
24 to shape when you stretch it by taking a breath  
25 because it's like a thick balloon, and this is just a

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1 moth-eaten tissue that has totally lost its structure  
2 and can't snap back, so it won't have any elasticity  
3 and it can't empty.

4 MR. CIRESI: Your Honor, could we have the  
5 clerk pass those.

6 (Clerk displays the exhibits to the  
7 jurors.)

8 Q. Doctor, in the course of your work, do you need  
9 to measure lung capacity?

10 A. Yes. I mentioned, you know, the chronic  
11 obstructive lung disease, the obstruction is the fact  
12 that the patient can't exhale, and how we determine  
13 if someone has it and how we determine how bad it is  
14 is by making direct measurements of how well the  
15 patient exhales air from their lungs. I told you  
16 that the basic problem was that if the person  
17 couldn't exhale, that they couldn't empty their lungs  
18 normally. And you have to sit and try to think how  
19 am I going to -- how am I going to measure that?  
20 Eventually, you know, you come up with the idea that  
21 you have the patient take a deep breath and blow out  
22 as fast as they could, and just measure in some way  
23 how fast the air would come out, and that would tell  
24 you if there's obstruction to air flow. And that's  
25 exactly what we do in the pulmonary lab, is the

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1 standard diagnostic test for finding out if you have  
2 airway obstruction and finding out how bad it is.

3 Q. And have you prepared an exhibit which would  
4 illustrate what is done, --

5 A. Yes, I have.

6 Q. -- and that's 30268?

7 A. Now --

8 MR. CIRESI: Your Honor, we would offer  
9 30268 for illustrative purposes.

10 MR. MONICA: No objection.

11 THE COURT: Court will receive 30268.

12 A. Okay. Now this is a graph, and first of all we  
13 start at the vertical axis of the graph, and you'll  
14 see it says volume, and we're measuring the volume of  
15 air that a person can breathe out and we're measuring  
16 it in liters, so that there's one, two, three, four  
17 and five liters of air. Someone can hold about five  
18 liters of air in their lungs. And that volume of air  
19 is measured as someone blows it out. And on the  
20 right you see it says time, and here in this graph  
21 the time is in seconds, one, two, three, four, five,  
22 six seconds.

23 And what we do is we take -- tell the patient,  
24 "I want you to take a deep breath, fill your lungs as  
25 full as you can with air, and then I want you to

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1 breathe it out as hard and as fast as you can until  
2 your lungs empty, till they're empty." And we  
3 measure exactly what happens to their volume.

4 And I want you to look only at the top curve for  
5 a second, and that's a normal what's called -- this  
6 is called the spiro -- spirogram -- spiro is just  
7 breathing, gram is a graph of breathing -- and the  
8 top line is what's called the normal spiogram. And  
9 when the patient -- what you can see is that when the  
10 patient breathes out, that little dot where it says  
11 FEV1 --

12 Q. What is FEV, doctor?

13 A. FEV1 means the --

14 The F just means that we ask the patient to  
15 breathe hard, that they forced their expiration. And  
16 E is just the exhaled volume. So it's the forced  
17 exhaled volume. The patient takes a big breath and  
18 they force it out as fast as they can with a forced  
19 exhalation. And the little one just means that that  
20 is how much air came out in one second. And that's  
21 why if you look where that dot is, it's right above  
22 the one second mark on the graph.

23 And what that shows you is that when a normal  
24 person breathes out hard, 80 percent of the air comes  
25 out in one second. His lungs -- they're stretched

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1 when you take a deep breath, they're really elastic

2 like a stiff balloon, and the pipes that conduct the  
3 air out them are held open and they're big enough to  
4 empty the lung. And so when you breathe out hard, 80  
5 percent of the air comes out in one second. The rest  
6 of the 20 percent comes out in another second or two.  
7 So within -- within two or three seconds all of your  
8 lung is empty and no more air is coming out, you're  
9 just going flat along that curve. So that's what  
10 normal lungs are supposed to do. You take a deep  
11 breath to the size of your lungs, and if you blow out  
12 hard -- you can all try this, you blow out hard and  
13 your lungs will be empty in just a second or two and  
14 there won't be any more air coming out.

15 And that's how we measure whether someone has  
16 COPD, is by doing a test like this.

17 Q. Now what is depicted with the second line, which  
18 is titled "Moderate Obstruction?"

19 A. Well the second line is someone who has COPD  
20 that's moderately severe, and by -- we mean  
21 moderately severe is it's gotten bad enough where in  
22 a normal-sized person only about one and a half to  
23 two liters of air will come out in one second. And  
24 that that reduction is due to the mechanisms that we  
25 talked about, the narrowing of the pipes, the

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1 dissolving of the tethering of the pipes, and then  
2 the loss of elasticity of the lung tissue itself.

3 And you can see several things about that curve  
4 that are different. First of all, if you go up on  
5 one second, only about one and a half liters came  
6 out, and that's only about a quarter of the air came  
7 out in the first second. Instead of 80 percent comes  
8 out in the first second, only a quarter of the air  
9 came out in the first second. And as long as the  
10 patient keeps breathing out and out and out, the lung  
11 keeps emptying and emptying and emptying. So it  
12 doesn't really get emptied, it just continues  
13 emptying until the patient has to stop and take  
14 another breath. So this is a lung clearly that can't  
15 empty very well. The patient cannot empty the lung.

16 Then the third graph is just -- it shows what  
17 someone with severe, terrible obstruction, and I want  
18 you to pay close attention so this because we're  
19 going to talk about a patient who has this -- has  
20 obstruction this severe and show you an illustration  
21 of this. But this is where the FEV1, the amount of  
22 air that comes out in one second here, is under one  
23 liter of air, so it's about maybe 600 -- .6 tenths of  
24 a liter, so it's maybe 20 -- you know, 10 or 20  
25 percent of the lung volume can come out in one

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1 second. And you can see that it's slow to come out  
2 all the way along the way. It's just the lung cannot  
3 empty, and just a little air comes out piece by piece  
4 by piece by piece rather than the lung just snapping  
5 empty and going right down to empty within two  
6 seconds. And that would be someone with very severe

7 obstruction.

8 And people with moderate obstruction and  
9 terrible obstruction, it's not just something you  
10 measure in the lab, it's something that translates  
11 into real symptoms that cause people real  
12 difficulties in -- in living their lives.

13 Q. How does this manifest itself in terms of a  
14 person breathing?

15 A. Well everybody is -- is different, but patients  
16 who have mild obstruction can normally do things  
17 pretty well in their day-to-day life. They couldn't  
18 run a marathon very well, they couldn't do highly  
19 athletic things, but they could walk from place to  
20 place and carry on a relatively sedentary job and do  
21 okay and not be very bad. When it gets down to  
22 moderate obstruction, patients begin to get shortness  
23 of breath that interferes with the heavier things  
24 they have to do in their day-to-day lives, like  
25 walking upstairs, they go to church and there's ten

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1 steps up, or if there's a little something unusual or  
2 if they have to -- they may be fine walking in from  
3 the car, but if they have to carry a 20-pound bag and  
4 they get breathless and short of breath and feel  
5 hungry for air.

6 And then when you get to the bottom, the  
7 terrible, when the FEV1 gets under a liter, that's  
8 where any little activity -- you know, even if the  
9 patient is okay at rest, any little activity, walking  
10 a few feet, saying eight words, have to take another  
11 breath. Doing -- you know, just conversing, walking  
12 a little bit, carrying a very light things, anything  
13 like that would make the person short of breath. And  
14 eventually they end up being in a wheelchair because  
15 people have to move them, or a little motorized cart,  
16 and they end up with oxygen. Most of the patients  
17 have oxygen who have FEV1s under one liter, they have  
18 severe obstruction, and they end up with really being  
19 very, very disabled. Disabled from work, but  
20 disabled also from normal human activities that  
21 they're required to have a high quality of life by  
22 the severity of the shortness of breath that they  
23 have.

24 Q. Doctor, when someone is breathing with moderate  
25 or -- or heavy obstruction or terrible obstruction,

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1 is their breathing pattern different than the normal  
2 person would have?

3 A. Well they breathe faster. There's a number of  
4 things that -- that they do. They breathe faster  
5 because they're short of breath and they're hungry  
6 for air, but they also tend to --

7 One of the problems is because the lung can't  
8 empty, when they're ready to take another breath  
9 there's already air in the lung. In fact the tubes  
10 have collapsed, so the only way you could get a  
11 breath is to take a breath on top of a partly-filled

12 lung already. So that they tend to -- the lung  
13 volume tends to walk up and up to a higher lung  
14 volume so they can keep some little amount of air  
15 going in.

16 When the lung gets up to a very high volume,  
17 which it has to be so you can get some emptying -- so  
18 the lung can't empty, so the next breath has to be on  
19 top of that, can't empty so the next breath has to be  
20 on top of that, you end up with a patient who is  
21 breathing way up high with big, inflated lungs, and  
22 the work of breathing at that mechanical place is  
23 very, very hard, and one of the reasons is that the  
24 diaphragm is the main breathing muscle, this is the  
25 big piston-like muscle that's right under the rib

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1 cage. When you breathe in you can see your belly  
2 contents will come out as the diaphragm comes down,  
3 and what happens is if your lungs get too big, then  
4 the diaphragm is totally flat at the beginning of the  
5 breath and can't help you, so your main breathing  
6 muscle, because of the mechanical problems of not --  
7 of lungs that can't empty, becomes absolutely unable  
8 to help you take the next breath. So all you have  
9 left are your muscles of your neck and your muscles  
10 of your upper chest. And you can sort of watch  
11 people who have very big barrel-chest, severe  
12 emphysema, and you'll watch them breathe and they use  
13 all these neck muscles to breathe, we call them  
14 accessory muscles, and all their chest muscles to  
15 breathe, and it's very, very hard work, very, very  
16 hard work to breathe when the lung is -- when the  
17 chest wall's expanded and the diaphragm is flat and  
18 won't work and they're having to use all these extra  
19 muscles to get a little air on top of these great big  
20 lungs that can't empty normally because of this  
21 problem.

22 So you'll see people breathing visibly, you can  
23 watch their neck muscles tighten as they sort of  
24 fight to take a -- to take a breath in, especially  
25 when they work, but sometimes even at rest.

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1 Q. And doctor, have you prepared another graph  
2 which shows the effect of smoking in COPD over a  
3 period of time?

4 A. Yes, I have. And I think what it --

5 I made another graph to show you sort of the  
6 natural history of COPD and to talk a little bit  
7 about how this happens to a person over a lifetime  
8 when they get obstruction of their air tubes, when  
9 they get this chronic permanent obstruction of their  
10 air tubes.

11 Q. Can you direct your attention to Exhibit 30059.  
12 Is that the illustrative exhibit that you have  
13 prepared, doctor?

14 A. Yes, that --

15 Yes, it is.

16 MR. CIRESI: Your Honor, we would offer

17 Exhibit 30059 for illustrative purposes.

18 MR. MONICA: No objection.

19 THE COURT: Court will receive 30059.

20 A. Just like before, I want to go over this graph  
21 and just set up the vertical and the horizontal axes.  
22 And you can see that the vertical axis is the same,  
23 it's volume, and it's volume in liters. And this is  
24 the volume of air that comes out in the first second  
25 on one of these tests, so this is the F -- the forced

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1 exhalation volume in one second, and one liter, two  
2 liters, three liters, four liters and five liters.  
3 And on the horizontal axis, just like before, we have  
4 time, but here instead of time in seconds I have time  
5 in years, and time over the whole lifetime of an  
6 individual. What happens to your breathing tests  
7 from the time you're 20 until the time that you're  
8 70? So it's timing that's marked off in decades of  
9 life on the bottom axis.

10 And if you just start at the -- like I told you  
11 before, look at the dotted lines crossing the graph,  
12 when obstruction to air flow gets bad enough where  
13 your FEV1, the amount you can get out is about one  
14 to -- and a half to two liters is where people begin  
15 to get shortness of breathe where they do anything  
16 unusual, so you can call that shortness of breath  
17 with moderate activity. And when you get down below  
18 a liter is when you have shortness of breath with any  
19 minimal activity where you're really breathless,  
20 where you're really disabled, where you face a risk  
21 of dying, where you get on chronic oxygen, where you  
22 struggle to breathe and fight to breathe even to do  
23 normal human activities of your life. So that's  
24 where the two liters is moderate obstruction and the  
25 one liter is severe obstruction.

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1 Now if you look at the very top line on here,  
2 you can see that a 20-year-old person has a FEV1 --  
3 and I'm using as -- as an example a 20-year-old male  
4 who is 70 inches tall, because that's the average  
5 height, and that person has an average FEV1 of four  
6 and a half liters. The F --

7 The total size of your lungs is determined by  
8 your body size, by your age and by your sex to a  
9 certain extent. So we're going to use an example of  
10 an average-sized male who is 20 years old. They can  
11 blow four and a half liters of air out of their lungs  
12 in one second, just like that -- exactly like the top  
13 line on the last graph that I showed you.

14 What happens if you take that person who doesn't  
15 smoke and has a normal life experience and follow  
16 their breathing tests throughout their whole life?  
17 And what you find is that there's a slow, gradual  
18 decline in the FEV1 of about 30 cc's per year.  
19 That's about like a shot glass smaller. So each year  
20 your breath -- the breath that you can force out of  
21 your lungs gets about one shot glass smaller over

22 your entire lifetime.  
23 Why is that? Part of it is due to your chest  
24 cage getting a little stiffer. It's due to  
25 mechanical things as your ribs and your articulation  
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1 of your ribs with your spine changes a little bit.  
2 Even changes in your spine. Part of it is due to the  
3 elastic tissue in your lungs gets less tensile, less  
4 elastic as you get older. There's physical/chemical  
5 changes in elastic tissue. So for whatever reason  
6 there's a small dropoff in your first-second volume  
7 that happens over your whole lifetime.

8 But you notice when you're at 70, you're still  
9 at three and a half or four liters, you still got  
10 double the breathing power that you would need  
11 to -- before you even get down into that beginning  
12 symptomatic range, and people live out their lives  
13 without ever being limited by their breathing power.  
14 Their lungs always empty well.

15 Now the second line shows you that a certain  
16 percentage of smokers, probably in the range of 15 to  
17 20 percent of smokers, are -- basically develop an  
18 accelerated loss of lung function over their entire  
19 lifetime, and those smokers -- what happens to them  
20 is that they begin to lose their lung volume faster.  
21 Their FEV1 drops about a hundred cc's a year, so  
22 roughly about three times faster. And that seems to  
23 occur from an early age, from 20 or even earlier.  
24 They -- when these subjects start to smoke, their  
25 breathing power starts to drop off faster and faster.

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1 And you can see that they're not going to run  
2 out of breathing power in their twenties, this is  
3 because these two curves are only separating at 50  
4 cc's a year, so it takes a long time and years and  
5 years of exposures, but they are losing lung function  
6 in an accelerated fashion right from the beginning.  
7 And what happens is if they continue to smoke and are  
8 susceptible to the effects of smoke, that they  
9 gradually in their early forties begin to get into  
10 the area of mild obstructive lung disease that we can  
11 measure and pick up with these tests.

12 By the time you follow this other line, when  
13 you -- by the time they hit the one liter mark, in  
14 other words, a large group of patients who have  
15 developed severe COPD, these are the patients that  
16 you see riding a cart, oxygen in their nose, unable  
17 to walk around the store, disabled people working  
18 hard to breathe, those type of patients have an  
19 average FEV1 of about a liter, and they reach that at  
20 an average age of about 58.

21 So that the average person with severe COPD is a  
22 58-year-old person who has smoked their whole  
23 lifetime and has had this gradual accelerated drop in  
24 lung function over their whole span of their -- of  
25 their smoking life. And by the time they're 58, they

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1 cross that line into severe respiratory disease,  
2 severe COPD.

3 Now what does that mean, to have an FEV1 of a  
4 liter when you're 58? Well different doctors have  
5 followed large groups of patients where they -- with  
6 chronic -- that's irreversible, they tried to treat  
7 all the reversible parts and what's left is chronic  
8 obstructive lung disease with an FEV1 of one liter  
9 about, and they followed them for years in studies  
10 and so to see what happens to these people. And when  
11 you pick up people right where that line crosses the  
12 second -- the bottom dotted line, 50 percent of them  
13 die of their lung disease within five years. So  
14 these patients are dying of their COPD in their early  
15 sixties, late fifties, early sixties to a large -- to  
16 large part.

17 This is a very severe disease which not only  
18 causes a lot of shortness of breath, suffering,  
19 distress in even doing day-to-day activities, but  
20 also leads you to lose your life early and to die  
21 of -- of -- a breathless death in your early sixties.  
22 And to lose out on that part of your life, your  
23 sixties and your seventies where you're retired and  
24 where your grandchildren are being born and -- and  
25 being raised, and it's -- it's a very tragic and

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1 unfortunate time point. Now --

2 Q. Doctor --

3 MR. MONICA: Excuse me.

4 Q. Doctor, when you --

5 MR. MONICA: Excuse me, Mr. Ciresi. Your  
6 Honor, I move that the -- at least the last portion  
7 of that answer be stricken as a gratuitous statement,  
8 comment by the doctor on philosophy of life or  
9 whatever it was. And also I would ask that counsel  
10 ask a question and answer instead of a running  
11 narrative, Your Honor. I object.

12 THE COURT: All right. Well the last  
13 portion was not responsive to the question.

14 Try and ask questions, counsel.

15 MR. CIRESI: All right.

16 BY MR. CIRESI:

17 Q. Doctor, when you get to the severe stage, will  
18 cessation of smoking help?

19 A. When you get down to the very severe stage where  
20 the FEV1 is a liter, cessation of smoking can help  
21 you have less secretions, can improve the quality of  
22 your life a little bit, but it does not change the  
23 gradual decrease in lung function and it does not  
24 change the mortality. So at that point there's some  
25 subjective benefits in terms of how much coughing and

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1 how much secretions and how many infections, but it  
2 does not alter the natural history in terms of the

3 progressive nature of the disease from that point or  
4 the dying of the patient from that point.  
5 Q. And the line that's sort of the broken line  
6 which says ex-smoker, can you describe what is being  
7 depicted there?  
8 A. Well that's one of the most important things, I  
9 think, of this whole area, is that if patients have  
10 obstructive -- chronic obstructive disease that's  
11 still moderate, then if at that point they quit  
12 smoking, it can make a drastic difference in the  
13 eventual outcome and the natural history of their  
14 disease. And that's what lung doctors like myself  
15 do, is to try to find people who are developing  
16 obstructive lung disease when it's still in its  
17 moderate stage when they're in their early forties  
18 and work with them to get them to quit smoking.  
19 And what you can see is on that line, there's a  
20 line where the hatch line goes off, and the patient  
21 is in their mid-forties and say the FEV1 is 22 -- 2.2  
22 liters or 2200 milliliters. What I use in talking to  
23 my patients and teaching my patients is I use how  
24 much left -- they have left to spend before they have  
25 severe COPD.

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1 So if you came in and you were 46 years old and  
2 you'd smoked since you were 15 and your FEV1 was 2.2  
3 liters, I would tell you you have 1200 cc's left to  
4 spend and -- before you develop this point of severe  
5 lung disease. If you continue to smoke during the  
6 next 10 or 15 years and you spend it at 100 cc's per  
7 year, you're going to reach that point in your mid-  
8 to late fifties and you're going to be in this group  
9 of people with severe symptoms and major disability.  
10 On the other hand, if you can quit, then you go  
11 back to losing it at 30 cc's a year. You don't get  
12 your lung function back, you don't jump up to that  
13 top line, but you go back to losing it at 30 cc's a  
14 year. And if you have 1200 to spend and you spend it  
15 at 30 cc's a year, that's going to last you decades,  
16 and the middle part of your life is going to have a  
17 different course than it will if you run out of  
18 breathing power when you hit that dotted line at --  
19 at -- in -- in your late fifties.  
20 Q. So that if they can quit, they will reduce  
21 the degenerative condition of the lung; is that  
22 right?  
23 A. They will slow the rate of decline of their lung  
24 function from the point where they quit, and it can  
25 make a drastic difference clinically.

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1 Q. Doctor, can you direct your attention to Exhibit  
2 30057. Is this an illustration of the type of  
3 breathing that you described a little bit earlier?  
4 A. Yes, it is.  
5 MR. CIRESI: Your Honor, for illustrative  
6 purposes we would offer Exhibit 30057.  
7 MR. MONICA: No objection.

8 THE COURT: Court will receive 30057 for  
9 illustrative purposes.  
10 BY MR. CIRESI:  
11 Q. Can you describe, doctor -- and we'll try to  
12 bring it up as you talk about different parts of the  
13 exhibit --  
14 A. All right.  
15 Q. -- can you describe what is being depicted here?  
16 A. Now this -- this is an illustration by a famous  
17 medical illustrator named Frank Netter, and he's  
18 drawing his perception of what someone with bad  
19 emphysema looks like as they're trying to breathe.  
20 On the top left you can see the schematic of the  
21 lung, shows the things that we've talked about. See  
22 those two arrows that are vertical in the middle are  
23 where the airways are narrowed so they can't empty,  
24 and then it shows the big air space with the positive  
25 dots in it, which is the emphysema, the sort of holes

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1 in the lung because the lung has been dissolved, and  
2 the loss of that tissue has -- gets rid of the little  
3 tethering structures that hold the airways open and  
4 loss of that tissue loses the elastic ability to  
5 empty the lung. So this lung can't empty for all  
6 those reasons. There's no recoil because the lung  
7 has been dissolved and is moth-eaten. The tethering  
8 part of the lung can't open the little airways and  
9 the little airways are scarred and narrowed. And  
10 this is showing the exact physiology that we talked  
11 about.

12 The reason that I wanted to put this in is that  
13 it shows a couple things. If you show me the picture  
14 of the patient -- the drawing rather. It shows, for  
15 one thing, that -- that this patient's lungs, because  
16 of these -- this obstruction, don't empty. He has  
17 chronic obstructive lung disease, irreversible,  
18 problem exhaling, and so he's trying to compensate  
19 and get as comfortable as he can to breathe, and one  
20 of the things is the very bottom where the diaphragm  
21 is, that big piston that moves air in, you can see  
22 that that's flat. Contracting that is not going to  
23 do anything for him. He can't get any more air in  
24 because that diaphragm is already totally flat. So  
25 what he does is lean forward and roll his shoulders

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1 forward a little bit and use these big muscles of his  
2 neck and his upper chest to get that little bit of  
3 air in on top of his lungs that can't empty, to get a  
4 little bit of air in on top. And you've seen people  
5 breathe like that.

6 The other thing he's doing is if you look at  
7 that top arrow pointing at his lips, one of the ways  
8 in which a patient like this can get a little more  
9 air out is actually make -- put some backpressure in  
10 into the airway. It tends to splint it open just a  
11 little bit so there can be more airflow. And on the  
12 bottom right, if you can go up again, the bottom

13 right just shows that schematically, is that here on  
14 the left that airway is totally collapsed and can't  
15 empty at all, and on the right there's a couple of  
16 positive plus signs in the airway that the patient  
17 has made a little backpressure that just keeps that  
18 last little airway from collapsing so he can get a  
19 little airway out.

20 And patients do this automatically. When they  
21 take a breath, they sort of give backpressure  
22 by (demonstrating by pursing lips) -- just to hold  
23 their lung open a little bit and splint these airways  
24 so it can empty. And you don't have to teach them to  
25 do that, although they do at certain breathing

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1 classes, but the patients will do that automatically.  
2 So if we go back up to the picture of the  
3 patient, you can see that patient is leaning forward,  
4 they don't have a diaphragm any more because their  
5 lungs are so inflated and because they can't empty,  
6 they're sucking -- they're basically using the  
7 anterior chest and their neck muscles to get that  
8 little piece of air in, and then to try to empty a  
9 little bit they're pursed-lip breathing, closing  
10 their lips and (demonstrating by pursing lips) to --  
11 to give back pressure.

12 We're going to show you a patient who is  
13 breathing, and I want you to pay attention during the  
14 video part to her lips and just watch how she  
15 automatically adjusts to this to try to get that  
16 little last little bit of air in. I don't want --

17 I want you to realize how much difficulty this  
18 is to breathe like this. One of the things is that  
19 the average size of these lungs is very near to  
20 what's called total lung capacity at the end of a  
21 breath. Okay? So that their lung capacity is nearly  
22 at total lung capacity at the end of a breath, and  
23 that means all their breathing has to be done on top  
24 of that. So it -- what -- if you do is you just take  
25 a very big breath until your lungs are totally full

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1 and then pretend you have to start your breath there,  
2 and if I do that, the only way I have to get a little  
3 more air in is to pull with my neck muscles and my  
4 upper chest, and that's it. You know, that's the  
5 only way to stay alive is to take that next breath  
6 way at the top using the accessory muscles, leaning  
7 forward, pressing the stomach in. And it's very much  
8 work, the patients get exhausted from breathing hard  
9 and doing this intense work of breathing.

10 And eventually these muscles fail and the  
11 patient ends up in the hospital on life support  
12 machines, being rested and being taken care of in  
13 trying to treat whatever little reversible piece,  
14 whatever little infection, whatever little  
15 bronchospasm there is.

16 Q. Now doctor you said -- you mentioned we have a  
17 video, and this is illustrative of this breathing

18 that you've been describing?  
19 A. Yes, it is. It shows -- just shows a patient  
20 with severe COPD and, you know, the kind of work it  
21 entails to just to regular day-to-day activities.  
22 Q. Can you describe the individual that we'll be  
23 seeing?  
24 A. The individual is a patient of mine that I've  
25 taken care of for 12 years, and she's 52 years old at  
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1 this time. She's a nurse. She started smoking  
2 cigarettes at age 13, was smoking a pack a day by age  
3 14, was -- and has never been able to quit despite  
4 really heroic attempts. She's used every resource in  
5 the community and every resource that a doctor could  
6 give her to help her to quit, including smoking  
7 sessions, smoking cessation classes, groups, nicotine  
8 gum, nicotine spray, nicotine patches --  
9 MR. MONICA: Excuse me, Your Honor. May I  
10 make an objection. I object to the witness carrying  
11 on this narrative and describing what his patient has  
12 done. This is rank hearsay, Your Honor, and it's a  
13 narrative form of hearsay by the witness, and I  
14 object to it.  
15 THE COURT: I don't see where this is  
16 hearsay. These are the doctor's observations.  
17 MR. CIRESI: That's correct, Your Honor.  
18 THE COURT: And I think they're  
19 appropriate. There should be a question directed to  
20 the doctor, however.  
21 MR. CIRESI: I will do that, Your Honor.  
22 BY MR. CIRESI:  
23 Q. Did the --  
24 Does this patient have any history of asthma or  
25 allergies?

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1 A. No.  
2 Q. How many times has she been hospitalized?  
3 A. Seven times, first time at about age 40 or 41,  
4 and in recent years about one time per year, usually  
5 for about a week and usually for a bad infection that  
6 just sort of tips her over the edge and tips her to  
7 the point where she needs help to stay alive.  
8 Q. And doctor, you said she was a nurse. Where did  
9 she work?  
10 A. At the University of Minnesota Hospital.  
11 Q. And what was her position there?  
12 A. She was a very senior --  
13 She was a very senior nurse supervisor, managing  
14 very a large sophisticated unit for a number of  
15 years. Had to take disability retirement in '96 at  
16 age 50.  
17 MR. CIRESI: Your Honor, we would offer,  
18 then, for illustrative purposes the video. It is  
19 Exhibit 30049.  
20 MR. MONICA: Your Honor, we object to the  
21 video. It is not a proper representation of what it  
22 purports to be, and in addition it's duplicative of

23 Exhibit 30057.

24 THE COURT: Court will receive 30049 for  
25 illustrative purposes only.

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1 (Videotape played.)

2 Q. Can you describe what we see.

3 A. There is a patient taking a treatment with a  
4 bronchodilator, with a little inhaler that makes a  
5 mist containing the medicine to open up the airways a  
6 little bit.

7 Now the the patient is just sitting, and you see  
8 there's oxygen in her nose and she has an oxygen  
9 tank.

10 And as she works harder doing some little  
11 activities, you'll see that she starts to breathe  
12 even more with her lips.

13 You note she has to stop on the way back up and  
14 then stop again in the garage to sort of catch up,  
15 coming up this little slope here, this driveway.

16 THE COURT: We'll recess for lunch,  
17 reconvene at 1:45.

18 THE CLERK: Court will recess, reconvene at  
19 1:45.

20 (Recess taken.)

21

22

23

24

25

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1 AFTERNOON SESSION.

2 THE CLERK: Please rise. The court is  
3 again in session.

4 (Jury enters the courtroom.)

5 THE CLERK: Please be seated.

6 MR. CIRESI: Thank you, Your Honor.

7 Good afternoon, ladies and gentlemen.

8 (Collective "Good afternoon.")

9 BY MR. CIRESI:

10 Q. Good afternoon, doctor.

11 A. Good afternoon.

12 Q. The patient that we saw in the video, was that  
13 her ordinary condition on a day-to-day basis?

14 A. I think one of the hard things about that tape  
15 is that that portrays her at a time when she was very  
16 good. You'd seen her taking a breathing treatment  
17 before to get every little bit of oxygen from the  
18 breathing treatment. She was wearing her oxygen.  
19 The secretions were white. She wasn't sick so that  
20 she needed to come to the hospital or anything else.  
21 That was during a time when she was really near her  
22 optimal good part of her range.

23 Q. Doctor, can you turn on your microphone. I  
24 think it may be off.

25 A. Excuse me.

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1 (Discussion off the record.)

2 BY MR. CIRESI:

3 Q. Now doctor, looking at the effect of chronic  
4 obstructive pulmonary disease from a medical  
5 standpoint, how many yearly United States  
6 hospitalizations are attributable to chronic  
7 obstructive pulmonary disease?

8 A. I think it's important to realize how very  
9 common a problem that this is, and it -- right now  
10 probably 10 to 15 percent of all hospitalizations,  
11 all hospitalizations in the United States are for  
12 treatment of this problem. It's a very, very common  
13 problem.

14 Q. And with regard to leading causes of death in  
15 the United States, where does it rank?

16 A. Right now COPD ranks as the fourth leading cause  
17 of death in the United States, behind heart disease,  
18 cancer, and I think cerebrovascular disease. But  
19 it's the fourth leading cause of death in the United  
20 States.

21 Q. And approximately how many people in the United  
22 States per year die from chronic obstructive  
23 pulmonary disease?

24 A. About -- approximately 100,000 deaths per year  
25 from this condition.

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1 Q. And what is the overwhelming cause of chronic  
2 obstructive pulmonary disease?

3 A. The overwhelming cause for -- for this condition  
4 is smoking cigarettes. It overwhelms all other  
5 causes.

6 Q. Are there any other causes, doctor?

7 A. There are other causes of COPD that do -- that  
8 do exist, and some of them are -- there's a very rare  
9 genetic disease where the patient has no -- has --  
10 misses an enzyme that protects you against the  
11 substances that dissolve the lung, and this is an  
12 anti-elastase. And that -- if you were born without  
13 that enzyme, then you get COPD at an early age. But  
14 even there smoking plays a dramatic effect on how  
15 fast you get it. If you don't smoke, about half of  
16 the people get short of breath by age 40 and about  
17 half of them have tied by age 55 of COPD, and this is  
18 non-smokers. If you smoke, half of them are short of  
19 breath by age 30 and half have died by age 40. So  
20 that even in that condition, smoking accelerates it.

21 And I think you have to put that in perspective  
22 because the incidence of that rare condition is about  
23 one in a hundred thousand patients, so if you look at  
24 the whole -- like the metropolitan area, there's 20  
25 or 30 patients with that condition. That's a rare

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1 condition. Whereas if you look at the number of  
2 patients with COPD, it's in the tens of thousands to  
3 upwards toward a hundred thousand. So that you're

4 talking about a rare genetic defect with 20 or 30  
5 patients in this metropolitan area versus something  
6 that is incredibly common as a clinical condition.  
7 Q. Are there any other causes other than this rare  
8 genetic disease and smoking?  
9 A. We've mentioned that the "chronic" part of  
10 chronic obstructive means the irreversible part, the  
11 damage to the lung that can't get better, and we  
12 talked briefly that asthma is an acute obstructive  
13 disease, that if you get the proper treatment, you'll  
14 go back to breathing normally and feeling normal.  
15 For example --

16 And that's what so, so terrible about this  
17 disease is it is like a asthma attack that isn't  
18 going to go away. No matter what you do, it's going  
19 to be with you like the patient that we watched. You  
20 just want to say "Well let me help you do that. Let  
21 me" -- you know, when you're watching her, you want  
22 to stop her and help.

23 And asthma ordinarily is very treatable, but  
24 there are some patients with asthma whose asthma  
25 condition, as they age and as they have more and more

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1 attacks, develops an irreversible part to the asthma  
2 so that the asthma itself becomes a chronic  
3 obstructive lung disease. But numerically those  
4 cases are very tiny; at most five -- five percent of  
5 the entire group of COPD.

6 Q. Now doctor, directing your attention to the  
7 medical treatment and medical management of COPD,  
8 have you put together a chart which categorizes  
9 medical management? And if you could direct your  
10 attention to 30060.

11 A. Yes, I have it.

12 MR. CIRESI: Your Honor, for illustrative  
13 purposes we'd offer Exhibit 30060.

14 MR. MONICA: No objection.

15 THE COURT: That's 30060, counsel?

16 MR. CIRESI: 30060.

17 THE COURT: That will be received for  
18 illustrative purposes.

19 A. This is just a list highlighting some general  
20 topics, more to keep me from forgetting certain  
21 things as I go through it.

22 What we're talking about here is the medical  
23 management of COPD, what can a -- can a doctor do for  
24 patients with this problem. We've talked --

25 Q. Doctor, let me ask you one thing first. Is the  
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1 management of the problem on an overall basis costly?

2 A. It's extremely costly.

3 Q. All right. Can we --

4 A. All parts of the treat -- all parts of the  
5 management are very expensive.

6 Q. Can you then start going through each part of  
7 the medical management regimen and describe each one  
8 as we go along.

9 A. Yes. Yes, I will.  
10 We've talked a little bit about the natural  
11 history and the effect of smoking cessation on the  
12 natural history. COPD is something that occurs in  
13 this group of smokers who start smoking early and  
14 over decades. They have an accelerated loss of lung  
15 function until in their late fifties they reach the  
16 severe point where they need a lot of treatment and a  
17 lot of support and have severe disability and  
18 shortness of breath.

19 We've talked about smoking cessation as being  
20 important, especially early on, at changing the slope  
21 of decline of breathing function, so that they can  
22 still have an adequate, functional life for years or  
23 even decades. And patients do a lot of things to try  
24 to quit smoking, a lot of which are very expensive.

25 They, you know, join self-help groups, they take

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1 nicotine replacement in various forms, gum, patches,  
2 and even a nicotine nose spray. You can see these in  
3 any kind of a drug store right on the counter next to  
4 the cigarettes. They're for sale without a  
5 prescription, over-the-counter, and they're very  
6 costly. They cost well over a hundred dollars a  
7 month, a hundred dollars a month to replace the  
8 nicotine.

9 There's also for smoking cessation a trial of  
10 using anti-depressant drugs which blunt the  
11 craving -- are believed to blunt the craving or help  
12 a bigger percentage of people quit, and those drugs  
13 are expensive.

14 And then some patients even go so far as to  
15 get -- do inpatient. Hazelton and the Mayo Clinic  
16 both offer inpatient treatment, quitting regimens,  
17 which no insurance pays for, so the patients have to  
18 pay for themselves. The program at Hazelton is  
19 several weeks long and it's about as much as if you  
20 went on a cruise. You know, that's what I tell my  
21 patients, spend the money there, see if you can get  
22 off rather than doing something enjoyable.

23 So a lot of money goes into smoking cessation,  
24 and a lot of doctors' visits and doctors' work goes  
25 to try to help people stop smoking.

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1 MR. MONICA: Your Honor, I object to the  
2 running narrative of the witness's answer. The  
3 answer was given in the first few sentences, and I  
4 ask that the witness be instructed to answer the  
5 question directly and to not ramble on. I realize he  
6 has to explain his answer, I have no problem with  
7 that, but these long, rambling answers, Your Honor, I  
8 object to them.

9 THE COURT: Okay. Ask another question,  
10 counsel.

11 MR. CIRESI: Yes.

12 BY MR. CIRESI:

13 Q. Now directing your attention to bronchodilators,

14 can you tell us what that is, sir?  
15 A. Bronchodilators are medicines that relax the  
16 smooth muscle part of the bronchiole wall, and  
17 they're very common treatment for asthma, and they  
18 treat the small asthmatic piece of COPD. A lot of  
19 patients with COPD have a little bit of spasm as part  
20 of the obstructive disease, those same small airways  
21 can have some spasm, but instead of like, for  
22 example, someone with bad asthma, their FEV1 might be  
23 one liter, and after full treatment it might go to  
24 four liters, so it would be completely reversible and  
25 the patient will go back to being normal. With COPD

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1 it might be one liter when they're sick and have an  
2 infection, and with all the extra bronchodilator  
3 therapy it might go to 1.3 liters. So they get like  
4 a 15, 20 percent improvement. Now that's not much,  
5 but sometimes it's crucial for them to be able to  
6 live their life and do their job and function.

7 So the bronchodilators are used and they're  
8 either inhaled -- little puffers like asthmatics use.  
9 You've seen people with little, you know, meter dose  
10 inhalers and they puff on that and it relieves  
11 asthma. In some cases they put a liquid right in a  
12 little mister, like you watch the patient use in the  
13 beginning of that video, and they breathe in over 10  
14 or 15 minutes to get a higher dose. And those  
15 bronchodilators cost in the order of probably a  
16 hundred dollars a month for this kind, and two  
17 hundred dollars a month for the inhaled kinds. And  
18 there's a number of different kinds that are -- with  
19 slightly different mechanisms, but they're basically  
20 asthma-type treatments.

21 Q. Okay. And doctor, the corticosteroids, can you  
22 describe that management.

23 A. Well drugs like cortisone are very powerful  
24 anti-inflammatory drugs and they're used for  
25 arthritis and they're used for lots and lots of

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1 conditions, and when people are really sick and  
2 almost dying with COPD, sometimes just reducing the  
3 inflammation in the lung can sort of get them over a  
4 very bad spot. Does not fix the lungs, but it can  
5 get them over a severe problem. So that oral  
6 steroids, like by pill, prednisone pills, are used to  
7 get them over very tough spots. And the inhaled  
8 cortisone on a chronic basis are sometimes used to  
9 try to alter the course. The trouble with inhaled --  
10 inhaled steroids is trying to reduce the inflammation  
11 and calm the lung down, and they're never as powerful  
12 as directly deeply inhaling something that causes  
13 inflammation 20 times a day.

14 So they do not counteract the effect of smoking,  
15 they are nowhere near that powerful, but they are  
16 used in this condition. And again, the cost of the  
17 inhaled steroids is like a hundred dollars a month.  
18 The oral steroids are cheaper, but -- by pill,

19 but they have more side effects.

20 Q. And doctor, antibiotics, how are they used in  
21 the medical management of COPD?

22 A. Well one of the things that gets people in  
23 trouble so they have to go into the hospital, like if  
24 they get the flu last month or this month, it's not  
25 just the fever and aching -- you know, aching and

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1 being sick for a few days, they also can't breathe,  
2 and then they can get a secondary infection with  
3 bacteria where they cough up green pus and they're  
4 infected. And they don't have enough reserve to have  
5 a bronchitis, so it sort of tips them over the edge  
6 when they have to go into the hospital. And when  
7 they get a bacterial infection in the bronchial tree  
8 or a bronchitis, they need antibiotics, and the  
9 antibiotics are expensive and have to be taken for  
10 episodes of bronchial infection.

11 Q. Are individuals who have COPD more susceptible  
12 to other illnesses or diseases?

13 A. Yes, they're -- they're certainly susceptible to  
14 either having the disease more often, like episodes  
15 of bronchitis, or not having the reserve so they get  
16 sicker and are in danger with the same kind of  
17 illness that someone with more reserve could do fine  
18 with.

19 Q. And doctor, how is oxygen used in the medical  
20 management of COPD?

21 A. Now oxygen is very important. And you saw her  
22 carrying her oxygen tank. And oxygen is one of the  
23 few treatments that actually has been shown to  
24 prolong life in severe COPD. And it only works for a  
25 small group, maybe a quarter of the patients, because

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1 not all patients with COPD have very low oxygen. But  
2 the ones that have very low oxygen, either at rest or  
3 with exercise, who use oxygen, it -- it basically has  
4 been shown to prolong their life by up to two or  
5 three years. So that all patients who have resting  
6 low oxygen or whose oxygen drops either at night or  
7 with exercise, we put on chronic oxygen, and they use  
8 that not only to be able to exercise better, to avoid  
9 turning blue when they try to exercise, but they also  
10 use it as an actual life-prolonging measure. And  
11 it -- it prolongs life on the average of -- of two to  
12 three years.

13 It costs two to four hundred dollars a month,  
14 depending on whether you have a concentrator, whether  
15 you have extra tanks that you carry around.

16 The patient in the video, when she -- her oxygen  
17 level was about 90, which is sort of the low end of  
18 normal, and when she would walk 50 feet it would drop  
19 below 80, which means blue basically, she would turn  
20 blue with walking 50 feet without oxygen. With the  
21 oxygen she could walk twice as far without dropping  
22 the oxygen level. So it did improve her capacity to  
23 exercise. And again what you saw was her exercise

24 with the help of oxygen, with the help of the  
25 bronchodilator.

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1 Q. And doctor, pulmonary rehabilitation, how is  
2 that utilized in the medical management of COPD?

3 A. Pulmonary rehabilitation is sort of a  
4 controversial subject, because it's not really proven  
5 that it prolongs anybody's life by going through  
6 different rehab programs, but it definitely makes  
7 people able to walk further and gives them a  
8 little -- it teaches them to be more relaxed with  
9 their breathing, what to do with they run out of  
10 breath, it teaches them how to do the pursed-lip  
11 breathing, make sure that they're using all their  
12 inhalers correctly. It's sort of a setup where they  
13 go through all the details of this chronic disease  
14 and try to optimally handle all of the details. Can  
15 either be done as an outpatient, and there's programs  
16 at most of the hospitals that run for like twice a  
17 week for six weeks or something like that. I use the  
18 program at Abbott Hospital quite a bit because it's  
19 right in the neighborhood of our hospital, and my  
20 patients, you know, do feel more confidence with  
21 their disease, learn about their disease, have a  
22 better functional life after going through that  
23 program. They don't live any longer.

24 And the other way you can do it is an inpatient  
25 program, and there are several hospitals in town

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1 including Vencor, which is the chronic subacute  
2 hospital that I work at that has an inpatient  
3 program, so people can actually go into the hospital  
4 for two weeks and get help with their secretions,  
5 with their bronchodilator use, with learning how to  
6 take care of their oxygen equipment, with pursed-lip  
7 breathing, with all the details of doing as well as  
8 they can with a chronic, incurable problem.

9 Q. Doctor, you talked about a chronic subacute  
10 hospital such as Vencor. What is a chronic subacute  
11 hospital?

12 A. Well it's a licensed --

13 Hospitals are usually licensed as acute  
14 hospitals, like the University of Minnesota, Mayo  
15 Clinic, St. Mary's down there, Hennepin County,  
16 Abbott, but there are a couple hospitals that -- that  
17 are licensed under a subacute hospital where the  
18 average length of stay, instead of being three or  
19 four or five days for acute problems, is more like 30  
20 or 40 days for things that sort of get better week by  
21 week and require a rehab component to it rather than  
22 the things that are getting better day-by-day. And  
23 in -- in the metro area there's two of those  
24 hospitals, ones in the west metro, in Golden Valley,  
25 and one's Bethesda, which is not too far from here,

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1 near downtown St. Paul.

2 Q. Doctor, hospitalization, is that required in the  
3 medical management of COPD?

4 A. Again it's 10 or 15 percent of all  
5 hospitalizations to acute hospitals in the United  
6 States, much higher percentage of hospitalizations in  
7 chronic long-term acute hospitals. So that many  
8 patients, especially when they get a bronchitis or an  
9 infection or some intercurrent problem, they have to  
10 go into the hospital, be stabilized and get their  
11 breathing back up to where it's good.

12 Q. What's the cost of hospital stays for COPD  
13 management?

14 A. Well they vary according to what needs to be  
15 done. But in general if the patient is in a ward bed  
16 getting an antibiotics and intensive treatment and  
17 things like that, the cost would be in the range of a  
18 thousand dollars a day. If they're in an ICU on a  
19 breathing machine, on life support, the cost would  
20 run from two to three thousand dollars a day. So  
21 that the -- you know, hospitalizations have become  
22 incredibly expensive, and even a week in the hospital  
23 to treat a bad infection associated with COPD would  
24 be ten thousand dollars, easily.

25 Q. All right. Now doctor, it's also mentioned here

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1 single lung transplant or lung reduction surgery.

2 Let's talk about the single lung transplant.

3 Can you describe that aspect of medical management of  
4 COPD.

5 A. Uh-huh. The two things on the right I put in  
6 parentheses because they're sort of surgical  
7 management rather than medical management, but there  
8 are a couple things that are done occasionally to try  
9 to help people who are near the end of their life and  
10 are desperately short of breath from COPD. And  
11 sometimes, like you say, if these lungs are so rotten  
12 and so worn out and so terrible, why don't we just  
13 give them a new lung, you know, new lungs? And that  
14 is done. There are certain number of people,  
15 especially people who are young, who don't have other  
16 diseases, you know, who have not had other chronic  
17 diseases, who get a lung transplant. And they go  
18 from not being able to breathe at all to being able  
19 to walk and live -- live their life and get a  
20 tremendous relief. So that for some of these  
21 patients, that can be almost a miraculous type of  
22 improvement.

23 Q. Are there many available lungs in the United  
24 States to do lung transplants?

25 A. The problem is partly in organ -- is in cost, in

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1 the complications, and the organ availability. There  
2 is at least probably eight to ten thousand people  
3 right now in the United States who would benefit from  
4 a lung if it were available, and there's only three

5 or four to five hundred a year that are available.  
6 So most patients simply can't have one because there  
7 aren't available lungs that can be used for  
8 transplantation.

9 Q. Is it an expensive procedure?

10 A. The cost of a lung transplantation in the first  
11 year would run over a hundred thousand dollars, in  
12 the range of a hundred thousand dollars.

13 And the patient -- it's really done for  
14 lifestyle reasons. The patient is so short of breath  
15 that each breath, they feel like they're suffocating,  
16 and they would do anything to get rid of that sense  
17 of dyspnea. Because a third of these patients die in  
18 the first three months of either rejections or of  
19 infections, so the patient that wants to go for lung  
20 transplant -- one, they have to get incredibly lucky,  
21 a lung has to become available when they need it, and  
22 they have to have a chance, a 30 percent chance of  
23 dying within months in order for -- you know, to take  
24 that chance for what's at the other end, is some  
25 patients who get relief of their symptoms.

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1 Q. Are there other sequelae or consequences of a  
2 lung transplant other than death?

3 A. Well a lot of people get rejection of the organ  
4 which causes obstruction itself and leads to a  
5 recurrence of the shortness of breath. And the  
6 patients on transplant -- on -- with lung transplants  
7 have to take high doses of immunosuppressive drugs,  
8 and they get infections from those drugs. And they  
9 have to take high doses of cortisone, which leads to  
10 softening their bones, fractures of their spines,  
11 necrosis of their hips, they need hip surgery in many  
12 cases, cataracts with cataract surgery, so that  
13 there's a whole complication of the regimen of drugs  
14 that you have to take to keep from rejecting that  
15 lung, and it's simply not an available treatment for  
16 the tens of thousands of patients who have COPD as  
17 they reach the end of their life. It's something  
18 that is done in a few -- a few patients.

19 Q. Doctor, with regard to lung reduction surgery,  
20 can you describe that, please.

21 A. Well that's sort of an interesting story, too.  
22 One of the problems that I mentioned to you is these  
23 lungs are just too big, and one of the consequences  
24 of being too big is the diaphragms, that big muscle  
25 that works as a piston for breathing, is flat and

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1 can't work, and so people have come up with the idea  
2 of removing part of the lung to make the lung smaller  
3 so the diaphragm can work again and so that gas  
4 exchange with improve, and they actually are helped  
5 in this by the fact that emphysema tends to be worse  
6 in the top of the lung than in the bottom. There's  
7 some regional differences in the disease. So they  
8 remove the top third of each lung. They do a surgery  
9 where they split the sternum, just like heart

10 surgery, and take out the top third of each lung and  
11 then -- then close the patient. And some patients  
12 get substantial relief of their shortness of breath,  
13 they double their ability to walk, and that lasts for  
14 up to a few years.

15 And that is surgery that's being evaluated to  
16 try to figure out which subgroup of these COPD  
17 patients have the most benefit and the least risk, so  
18 we can pick out people that actually help them with  
19 that treatment.

20 Q. And doctor, is that an expensive treatment?

21 A. I would guess that the minimal cost for that  
22 would be in the range of \$20,000. And if you get  
23 persistent air leaks or other surgical complications,  
24 it can run into ten of -- many tens of thousands of  
25 dollars.

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1 Q. Doctor, do some of the COPD patients require  
2 permanent life support?

3 A. Now some patients when -- when they have a bad  
4 problem and they have an acute worsening of their  
5 disease, they would have to go on life support with a  
6 breathing machine. And they either have a tube down  
7 their nose into their lung or in their mouth into  
8 their lung, or sometimes even a tracheostomy so that  
9 they can be put on assisted ventilation with a  
10 breathing machine. And hopefully they will get  
11 better from their infection, whatever caused them to  
12 worsen suddenly will be treated and they will be able  
13 to come off that breathing machine.

14 Sometimes it happens when they get influenza.  
15 They have bad COPD and they get influenza, and they  
16 can't live without at least temporarily being  
17 supported with life support.

18 Q. Is that treatment expensive?

19 A. That treatment is -- you know, during the time  
20 that you need it, is thousands of dollars a day. And  
21 the problem is that some people never get well enough  
22 to come off of that breathing machine, and then they  
23 sort of have to make the decision whether to come off  
24 and be helped to be comfortable with drugs like  
25 morphine, or whether to try to make their life as a

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1 disabled person as well as they can with chronic life  
2 support. If they get to that point, then the costs  
3 are tens of thousands of dollars a month for that  
4 type of support.

5 Q. Doctor, if smoking were eliminated as a cause of  
6 COPD, would we see a reduction of this illness in the  
7 United States?

8 A. If -- if smoking would gradually disappear and  
9 there were no smoking in our culture, in one  
10 generation COPD would be a rare condition. It would  
11 be something that most doctors didn't see, it would  
12 be something that most families didn't have patients  
13 or relatives -- it would be an unusual, rare  
14 condition rather than the fourth leading cause of

15 death in the United States and the cause of 10 to 15  
16 percent of all hospitalizations in the United States.  
17 It would be that dramatic an impact on the incidence  
18 of COPD.

19 MR. CIRESI: Thank you, doctor. I have no  
20 further questions.

21 MR. MONICA: Your Honor, may we have a few  
22 minutes to move the Elmo over here?

23 THE COURT: Let's take a short recess.

24 (Recess taken.)

25 THE CLERK: All rise. Court is again in  
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1 session.

2 (Jury enters the courtroom.)

3 THE CLERK: Please be seated.

4 THE COURT: Counsel.

5 MR. MONICA: May it please the court.

6 Good afternoon.

7 (Collective "Good afternoon.")

8 CROSS-EXAMINATION

9 BY MR. MONICA:

10 Q. Good afternoon, doctor.

11 A. Hello.

12 Q. Doctor, my name is John Monica and I represent  
13 Lorillard Tobacco Company, and I'm going to be asking  
14 you some questions this afternoon, not only on behalf  
15 of my company, but the other companies, as we have  
16 a -- the court has asked us to just have one attorney  
17 ask questions of every witness, and we're trying to  
18 do that. And so I'll be the attorney who will ask  
19 you questions this afternoon.

20 If you don't understand my questions, please  
21 tell me. I'll be glad to repeat them.

22 A. Okay.

23 Q. Doctor, chronic obstructive pulmonary disease,  
24 is it correct that that disease consists of mainly  
25 two subparts, emphysema and chronic bronchitis?

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1 A. The -- the -- it's kind of a -- of a clumsy term  
2 because it incorporates two main things, emphysema,  
3 which is a pathological description, and chronic  
4 bronchitis, which is a clinical description regarding  
5 cough and sputum production. So that --

6 But the reason that it's used so often is that  
7 it represents the changes in the lung mainly due  
8 to -- to cigarette smoking and almost nobody has pure  
9 emphysema or pure chronic bronchitis. So that most  
10 patients have features of both of these. The airways  
11 are -- have some changes and the lung tissue has been  
12 dissolved and they have a mixture of two, so rather  
13 than trying to divide a group right in half into  
14 emphysema and bronchitis, which really doesn't divide  
15 because most patients have features of both, this is  
16 sort of a lumping term that's used to describe  
17 patients with -- who have to have obstruction, they  
18 have to be trouble exhaling, and it has to be  
19 irreversible.

20 Q. But there are gradations in between, but  
21 basically at the one end and at the other you have  
22 emphysema and chronic bronchitis; right?  
23 MR. CIRESI: Objection, asked and answered.  
24 THE COURT: No, you may answer that. You  
25 may answer.

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1 A. As I said, most patients have some of the  
2 clinical features which would be described as chronic  
3 bronchitis, some of the anatomic features which would  
4 be emphysema, and they have features of both of  
5 these. So that at the very far end would there be  
6 someone with pure emphysema and someone with pure  
7 bronchitis? There could be someone whose clinical  
8 picture fit more closely pure emphysema and whose  
9 clinical picture fit more purely with the bronchitic,  
10 but they would be uncommon.

11 Q. And to -- and to determine where they  
12 are -- where the patient is at on the spectrum, you  
13 of course would have to review that patient's chart  
14 and medical records and talk with the patient; isn't  
15 that correct?

16 A. You can't really totally determine where someone  
17 is on that record because emphysema is a pathological  
18 diagnosis, and most of the time our diagnosis is made  
19 by that spirogram where we do breathing tests, and  
20 what we're measuring is actually the degree of  
21 obstruction, and we don't get accurate --

22 So we would never think in terms of putting  
23 someone along this -- the area. We would say they  
24 have COPD and this is how bad their obstruction is  
25 based on the -- the breathing test.

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1 Q. Okay. And by "COPD," we're talking about the  
2 disease that we've been -- you've been testifying  
3 about today; correct?

4 A. Yes.

5 Q. That's the shorthand term for it, "COPD;"  
6 correct?

7 A. That --

8 Yeah, that's the term we spent a lot of time  
9 trying to explain.

10 Q. Yes.

11 Now let's try to put things in perspective.  
12 Isn't it true, doctor, that if you take lifetime  
13 smokers, people who have smoked for their entire  
14 lives, that only 10 to 15 percent of lifetime smokers  
15 develop symptomatic COPD? Isn't that true, doctor?

16 A. About 15 percent of people with -- who are  
17 lifetime smokers develop symptomatic COPD.

18 Q. And by the same token, conversely, that means  
19 that 85 to 90 percent of smokers do not ever develop  
20 symptomatic COPD; isn't that correct?

21 A. That's correct to a point, although the -- that  
22 doesn't mean they have normal breathing tests and  
23 that they haven't developed some degree of airway  
24 obstruction. That has never been shown, what

25 percentage have absolutely normal lung function. But  
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1 there are a substantial majority who never get to the  
2 point of being disabled by the airflow obstruction.  
3 Q. In fact, it's more than a substantial majority,  
4 it's the vast majority. It's 85 to 90 percent.  
5 A. It's about 85 percent.  
6 Q. Yes, sir. And as far as the disease COPD, I  
7 believe you said about 10 percent of your inpatients  
8 you treat for COPD, about 10 percent of your patients  
9 have COPD that requires treatment in a hospital; is  
10 that correct?  
11 A. I didn't say that, no.  
12 Q. Well is --  
13 Let me ask you the question, then, directly.  
14 A. Uh-huh.  
15 Q. Isn't it true, doctor, that of your practice,  
16 only about 10 percent of your COPD patients require  
17 hospitalization?  
18 A. I didn't -- that's --  
19 I didn't mean to say that or imply that at all.  
20 I -- I stated that about 10 to 15 percent of all  
21 hospital admissions in the United States are due to  
22 COPD, but I don't have any number for the percentage  
23 of my own patients with COPD who are ever in a  
24 hospital. It would be -- I don't have -- have that  
25 accurate number.

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1 Q. All right. So you don't know what percentage of  
2 your patients require hospitalization --  
3 A. No.  
4 A. -- because of COPD; do you?  
5 A. No.  
6 Q. And you talked about some of the cost figures  
7 and in that regard, the last exhibit we had, which is  
8 Exhibit 30060 -- and I'm going to put this up just to  
9 remind you of which one I'm talking about, and  
10 I've -- I've written on mine here, but this is the  
11 exhibit I'm talking about, doctor. You went through  
12 some cost estimates, do you recall, just a few  
13 moments ago on that?  
14 A. Yes, I did.  
15 Q. I'm going to be talking to you about some of  
16 those cost estimates. But have you been advised,  
17 doctor, that the plaintiffs in this lawsuit are  
18 seeking reimbursement for costs that they spent to  
19 treat some of the patients who have COPD?  
20 MR. CIRESI: Objection, Your Honor, that's  
21 outside the scope of this witness's testimony.  
22 MR. MONICA: Your Honor, this clearly is --  
23 He's talking about costs with regard to this  
24 lawsuit, costs for treatment of patients that are  
25 relevant to this lawsuit.

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1 THE COURT: Counsel, I don't recall any  
2 testimony with regard to costs for this lawsuit. I  
3 think you can inquire about his statement on costs  
4 generally.

5 MR. MONICA: Okay.

6 Q. Doctor, with regard to the costs that you have  
7 estimated here, isn't it true that you did not make  
8 any kind of a particular study of the costs? These  
9 are just your general experience, your general  
10 impression?

11 A. I did make a few phone calls to our pharmacy and  
12 ask them how they price out these items, how much one  
13 inhaler costs. I called an oxygen -- one of the  
14 biggest oxygen vendors. And I have very personal  
15 experience of hospital charges and costs from some of  
16 the administrative things I did. So I never meant to  
17 present it to you as a study of any kind, but I think  
18 it's an accurate estimate of some of the costs that  
19 are involved in these -- in these items.

20 Q. And in fact, doctor, as you said, it was not a  
21 detailed study or any kind of a study on your part;  
22 was it?

23 A. It was -- it was not a study, no.

24 Q. It was kind of based upon your experience and a  
25 few conversations you had.

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1 A. I stated what it was based upon.

2 Q. And doctor, let's see if we can establish the  
3 extent of what you did do.

4 Now bear with me, I'm not an artist, but I  
5 thought it might be helpful just to put this up. I'm  
6 going to try to draw the state of Minnesota here  
7 first, doctor.

8 MR. CIRESI: Is it all right if I look at  
9 this one, Your Honor?

10 THE COURT: If you want to.

11 MR. CIRESI: We'll see if he's as good as  
12 Mr. Bernick.

13 MR. MONICA: I make no representations.

14 THE COURT: I think we're going to bring  
15 Mr. Bernick back.

16 (Laughter.)

17 THE WITNESS: There's a little bump on the  
18 top.

19 MR. MONICA: That almost looks like Texas.  
20 But I --

21 MR. CIRESI: We can tell you're from Kansas  
22 City.

23 MR. MONICA: I think the -- we can go  
24 through the points I wanted to make, even though that  
25 is a very crude drawing, I admit.

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1 Q. But anyway, this is the Twin Cities area  
2 right -- right here, doctor.

3 A. Yes, it is.

4 Q. Now you --

5 When you were making your inquiries, you -- for

6 example, you didn't check with anyone up here in the  
7 Moorhead Fargo area; did you?  
8 A. No.  
9 Maybe put Willmar on there, which is halfway to  
10 the South Dakota border straight left. I do outreach  
11 there one day a month for several years, and I -- I  
12 did talk to some of -- their pharmacy when I was out  
13 there. So I have looked at a little at outstate.  
14 I'm also from a small town, and I have a family who  
15 use some of these products, so --  
16 Q. You didn't check with anyone up in International  
17 Falls; did you?  
18 A. No, not up in that bump.  
19 Q. And how about over here by the lake, Duluth.  
20 You didn't --  
21 A. No, I did not check Duluth.  
22 Q. Okay. So my point is, as far as the studies --  
23 or the inquiries that you made, they were fairly well  
24 localized on the Twin Cities area with a couple of  
25 outlying. Is that a fair statement, doctor?

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1 A. That's a fair statement, sure.  
2 Q. And so your --  
3 And admittedly this is a crude drawing, but  
4 your -- your inquiries and the cost estimates, then,  
5 are really only really relevant and pertinent to the  
6 Twin Cities area; aren't they, doctor? They're not a  
7 state-wide, they don't purport to be a state-wide  
8 figure.  
9 A. No, this is true, but in all fairness, like drug  
10 stores are becoming more of a national chain, and if  
11 I buy an Albuterol inhaler in a Snyder's store in  
12 Minneapolis and in Duluth and in Rochester and in  
13 Albert Lea, they may be off by a few pennies, but  
14 they're not off by an order of magnitude. These are  
15 products that there's a profit margin on. Just like  
16 cigarettes wouldn't be double the cost in the Twin  
17 Cities as in Willmar, they would be roughly in the  
18 same general frame because they're a product with a  
19 profit margin.  
20 Q. But doctor, you -- as you said, you didn't  
21 actually check in these areas that I've mentioned to  
22 see how close the prices were; did you?  
23 A. That is correct.  
24 MR. CIRESI: Objection, asked and answered,  
25 Your Honor.

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1 THE COURT: It's been asked and answered  
2 now.  
3 BY MR. MONICA:  
4 Q. Now in -- in determining whether a cost, a  
5 particular cost is -- is reasonable, doctor, don't  
6 you have to look at what was done for that particular  
7 charge?  
8 A. I'm not --  
9 I don't exactly know what -- what you mean.  
10 Where --

11 Q. I'll try to rephrase.  
12 A. Maybe you can use an example.  
13 Q. Well to -- let's take an example. Let's, for  
14 example, look at a bronchodilator.  
15 A. All right.  
16 Q. To know if that charge is -- is appropriate,  
17 wouldn't you have to know if the procedure was really  
18 necessary and appropriate?  
19 A. You would have to assume that the patient had  
20 some benefit from the treatment. Is that what you're  
21 asking?  
22 Q. And not all patients use bronchodilators; do  
23 they?  
24 A. Not all patients do use them.  
25 Q. So you'd have to consider whether the individual

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1 patient really needed a bronchodilator before you  
2 would know if that cost --  
3 A. Right.  
4 Q. -- for that dilator was reasonable, wouldn't  
5 you, sir?  
6 A. Right. And that would be determined by the  
7 individual doctor working with that patient, testing  
8 them, and the patient -- to see if they had an  
9 appropriate clinical response to that drug so that  
10 the benefit to them was worthwhile.  
11 Q. And doctor, is the same true for all of these  
12 entries on this chart that we have on the -- on the  
13 screen here?  
14 A. There's --  
15 Q. You got --  
16 A. -- some exceptions. For example, oxygen, it's  
17 federally paid for -- I mean the federal payors, like  
18 Medicare, you're required to document very precisely  
19 what the oxygen level is in the blood. So in order  
20 to give oxygen, it's not -- I can't give you oxygen  
21 because I think you might feel better. I'm required  
22 to show what your PO2 level is or what it does with  
23 exercise. There's very stringent criteria for that  
24 oxygen and you can't have it unless you meet very  
25 uniform standard criteria where it's been shown to do

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1 you some good.  
2 The oxygen vendor that we use actually started  
3 in Duluth, it's pretty much become a -- it's  
4 Arrowhealth, and they -- you know, a lot of the  
5 companies have amalgamated, and so I think the oxygen  
6 costs throughout the state would be pretty close.  
7 And certainly you just can't have a doctor decide to  
8 give oxygen. It doesn't work that way. You have to  
9 say what are the criteria? Well it's a PO2 less than  
10 60, and you have to show that. The lab that does the  
11 blood test has to be certified and you have to  
12 present that documentation at the time that you give  
13 the oxygen. So I would assume that at least for  
14 oxygen, everybody getting it would -- would be --  
15 represent people who would benefit from it.

16 Q. Yes. But on the other ones that we've talked  
17 about, the bronchodilators, the antibiotics, the  
18 pulmonary rehabilitation, you'd have to -- to take a  
19 look at the individual patient and his chart or her  
20 chart to determine whether the cost was necessary and  
21 appropriate; wouldn't you, doctor?

22 A. A doctor would have to be ordering the treatment  
23 for an appropriate -- for an appropriate patient.  
24 That's -- that would be true.

25 Q. And in fact, in order to determine if these

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1 treatments are necessary, you would want to know, for  
2 example, whether a patient had a history of exposure  
3 to environmental -- certain undesirable environmental  
4 exposures; wouldn't you, doctor? Like say they  
5 worked in the coal mine or something like that, you'd  
6 want to know that; wouldn't you?

7 MR. CIRESI: Your Honor, I'm going to  
8 object to that as being outside the scope. It's also  
9 in improper form.

10 THE COURT: Sustained.

11 BY MR. MONICA:

12 Q. Well doctor, when you are evaluating a patient  
13 to determine if that patient has COPD, what are some  
14 of the things that you ask the patient? When they --  
15 when they come in to sit down with you for the first  
16 time, what are some of the things you ask the patient  
17 about?

18 MR. CIRESI: Objection, Your Honor, outside  
19 the scope.

20 THE COURT: No, you can answer that.

21 A. Uh-huh. Well a patient often comes with a  
22 particular problem. They have a presenting  
23 complaint, there's something -- some reason that  
24 they're going to the doctor. And if you talk -- so  
25 the patient might make an appointment because they

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1 have a complaint, something that they're troubled  
2 with. Usually that would be shortness of breath on  
3 exertion. They've noticed that when they try to do  
4 heavy work or carry something or do more work, that  
5 they would be short of breath.

6 The other main symptom would be cough, that they  
7 have developed a chronic cough, that they cough up  
8 phlegm from their chest and they have a deep, barky  
9 cough that persists and just won't go away like a  
10 cold would, and they're concerned about that. So  
11 those would be the two main symptoms.

12 Sometimes the patient is just there for a  
13 routine exam, and you might question whether they had  
14 COPD based on their physical exam, their pattern of  
15 breathing, observing them doing the things you do in  
16 giving a regular physical examination.

17 Q. And doctor, you would ask the patient for the  
18 family background; wouldn't you?

19 A. If the patient came in with a complaint, like on  
20 a complaint-centered visit, I might not. But if this

21 was my patient, I would have as part of their medical  
22 record a complete family history, social history, and  
23 a complete review of systems. If I was seeing a new  
24 patient that was coming to me for the first time,  
25 then I would get that information and it would be

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1 part of -- of their record.

2 Q. And you would need that information to diagnose  
3 the patient; correct? That's why you ask the patient  
4 for the information.

5 A. I would need that -- that information to fully  
6 understand that person and their health history and  
7 their various problems.

8 Q. For example, you'd want to know if the patient  
9 had HIV; wouldn't you?

10 A. I would -- if it --

11 I would certainly want to know if the patient  
12 had a history of -- of being exposed, having risk  
13 factors, having been documented to have HIV. But to  
14 my knowledge, that has nothing to do with the  
15 question of COPD.

16 Q. Don't people who have HIV get pneumonia and  
17 other respiratory illnesses much more frequently?

18 A. Patients with HIV do not get COPD.

19 Q. Don't --

20 Would you answer my question? Do they get  
21 pneumonia more frequently, Your Honor -- ah --

22 A. Patients are susceptible to a variety of bad  
23 infections. One of the problems is that their immune  
24 system is wiped out, so they get a whole variety of  
25 respiratory and other infections which have to be

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1 managed as part of a chronic illness, but --

2 Q. Which if --

3 A. -- it has nothing to do with COPD.

4 Q. Which if they are not managed properly can move  
5 into COPD.

6 A. No.

7 Q. Wouldn't you want to know if -- if your patient  
8 had worked in a factory and had worked grinding fiber  
9 glass every day? Would you want to know that?

10 A. I would want to know what work my patients have  
11 done, yes.

12 Q. And wouldn't you want to know if your patient  
13 had worked in a paint factory where the paint was in  
14 the air all day and they had breathed the air all day  
15 every day while they worked at that paint factory,  
16 wouldn't you want to know that?

17 A. I would want to know what occupations my  
18 patients have -- have done, what they've done for  
19 work.

20 Q. And doctor, did -- did you know that we've taken  
21 some depositions in this case of Medicaid recipients,  
22 and that one woman that I deposed myself has had all  
23 these experiences I just mentioned to you?

24 MR. CIRESI: Excuse me, Your Honor, I'm  
25 going to object. And I'm sorry to interrupt,

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1 counsel, but he knows that's an inappropriate  
2 question. It's already been ruled on.

3 THE COURT: Counsel, that is an  
4 inappropriate question, that has been ruled on in the  
5 past. Move on.

6 MR. MONICA: All right.

7 BY MR. MONICA:

8 Q. Doctor, have you examined the individual medical  
9 records of any of the Medicaid recipients for whom  
10 plaintiffs claim damages in this lawsuit?

11 A. I don't know who those claimants are. All I can  
12 say is that they -- I've taken care of patients every  
13 day of my work life for 19 years, and I've certainly  
14 represented -- I've certainly taken care of many  
15 patients who are receiving Medicaid within the state  
16 of Minnesota. So again, I don't know the parameters  
17 of -- of the details.

18 Q. And -- and so then, obviously, doctor, you're  
19 not in a position to say if any of those people have  
20 COPD because they smoked; are you, doctor?

21 A. I don't know --

22 I do not have a list of any of the patients  
23 you're talking about. I have no personal knowledge  
24 of any such a list.

25 Q. And by the same token, doctor, you're not in a

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1 position to say that any of the medical costs  
2 incurred by those patients is reasonable and  
3 necessary; are you, doctor?

4 MR. CIRESI: Objection, Your Honor, there's  
5 no foundation for that.

6 THE COURT: Sustained.

7 Q. Doctor, when you look at a patient for the first  
8 time, you said you're looking for -- you're looking  
9 for two things, shortness of breath and -- what was  
10 the other one, doctor?

11 A. I said when a patient comes to me with a  
12 complaint, and they come in because they have a  
13 patient-centered complaint, that the two most common  
14 ones would be shortness of breath and a cough,  
15 chronic cough.

16 Q. And --

17 A. That's what the patient -- what the patient  
18 would bring to me as a complaint.

19 Q. And if they had those two symptoms, then, you  
20 would want to investigate whether or not they were  
21 being caused by COPD; correct?

22 A. Yes.

23 Q. And by the same token, those same two symptoms  
24 can be manifested as a result of other diseases, non-  
25 COPD diseases; right?

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1 A. Yes.

2 Q. And what are some of the other diseases that  
3 manifest those two symptoms, non-COPD diseases?  
4 A. Well asthma could present with either shortness  
5 of breath on exertion or with cough, and various  
6 types of diseases of the lung tissue could present  
7 with shortness of breath and/or cough, like pulmonary  
8 fibrosis or sarcoidosis. In fact any lung  
9 disease -- the lung doesn't have too many ways that  
10 it can tell you that it's going bad, and almost any  
11 lung disease can present with shortness of breath  
12 or with -- or with cough.  
13 Q. So you'd have to investigate those various  
14 possibilities in order to determine whether it was  
15 COPD you're looking at or one of those other  
16 diseases; right?  
17 A. That's -- that is my job.  
18 Q. And again you'd have to look at the patient's  
19 chart and the medical history and things like that.  
20 A. Uh-huh.  
21 Q. Correct, doctor?  
22 A. That would be part of the information I would  
23 gather.  
24 Q. All right. And as you said, you haven't done  
25 that for any patient in this litigation, any Medicaid

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1 patient.  
2 MR. CIRESI: Objection, asked --  
3 A. I don't know if that's true or not.  
4 MR. CIRESI: Excuse me, doctor. Objection,  
5 it's been asked and answered.  
6 THE COURT: It's been asked and answered.  
7 Q. Doctor, have you talked with any of the damages  
8 experts in this case and given your input to any of  
9 them?  
10 A. No.  
11 Q. So you're not testifying on any element of  
12 damages in this case, to your knowledge?  
13 A. I'm testifying about COPD as one of the severe  
14 medical conditions caused by chronic smoking and  
15 about the medical management of -- of that condition.  
16 Q. But as far as you know, doctor, your work is not  
17 being used in any regard to compute damages.  
18 A. To my knowledge, it is not.  
19 MR. MONICA: I have no further questions.  
20 MR. CIRESI: I have no further questions,  
21 Your Honor. Thank you, doctor.  
22 THE COURT: You may step down.  
23 MR. CIRESI: Your Honor, we need to --  
24 THE COURT: Side bar.  
25

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1 (Side-bar discussion at follows:)  
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(Side-bar discussion concluded.)

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1 MS. NELSON: Your Honor, plaintiffs call  
2 Dr. Kenneth Graham to the stand.  
3 (Witness sworn.)  
4 THE CLERK: Please state your name and  
5 spell your last name for the record.  
6 THE WITNESS: Dr. Kevin J. Graham,  
7 G-r-a-h-a-m.  
8 THE CLERK: Please be seated.  
9 THE WITNESS: Thank you.  
10 MS. NELSON: Good afternoon, ladies and  
11 gentlemen.  
12 KEVIN J. GRAHAM  
13 called as a witness, being first duly  
14 sworn, was examined and testified as  
15 follows:  
16 DIRECT EXAMINATION

17 BY MS. NELSON:  
18 Q. Good afternoon, Dr. Graham.  
19 A. Good afternoon.  
20 Q. Dr. Graham, is your mike on?  
21 A. Is it on?  
22 Q. Yes. Okay.  
23 Dr. Graham, would you please briefly explain the  
24 expertise you bring to the court and the jury today.  
25 A. What my purpose in appearing today to the court  
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1 is to talk about cardiovascular disease and the  
2 clinical presentation of cardiovascular disease,  
3 specifically coronary artery disease or the  
4 development and presentation, clinical presentation  
5 of blockages in the coronary arteries of the heart,  
6 stroke and the loss of blood in some way to the  
7 brain, and peripheral vascular disease or decrease in  
8 blood flow to the extremities.  
9 Q. And when you say "clinical presentation," what  
10 do you mean by the word "clinical?"  
11 A. Much like Dr. Davies, I am a clinician, and  
12 every day when I get up it's my job to go and see  
13 patients, and that's -- so we deal with patients who  
14 present with clinical problems, or hopefully to  
15 prevent those clinical problems.  
16 Q. Dr. Graham, before we get into the substance of  
17 your testimony, I'd like just to take a moment to  
18 review with you your education and your training.  
19 It appears that you graduated from the  
20 University of Minnesota Medical School in 1981; is  
21 that correct?  
22 A. Yes, ma'am.  
23 Q. And in 1985 you completed an internal medicine  
24 residency at Hennepin County Medical Center; is that  
25 correct?

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1 A. Yes, ma'am.  
2 Q. Could you just briefly describe to the jury what  
3 a residency is in internal medicine?  
4 A. After the completion -- successful completion of  
5 medical school, there is a competitive arrangement to  
6 go to top internal medicine residency programs.  
7 Through the internal medicine program you learn  
8 essentially all the inpatient and outpatient  
9 modalities of the treatment of the adult patient.  
10 Q. And then you were awarded a cardiology  
11 fellowship at the University of Minnesota; is that  
12 correct?  
13 A. Yes, ma'am.  
14 Q. And you completed that fellowship in 1988.  
15 A. Yes, ma'am.  
16 Q. So that was a three-year post-residency  
17 fellowship.  
18 A. Yes, ma'am.  
19 Q. Could you explain to the jury what the medical  
20 field of cardiology involves.  
21 A. After a three-year residency, learning the

22 spectrum of adult medicine and the various  
23 specialties within that, touching each of those, a  
24 cardiology subspecialty fellowship concentrates on  
25 diseases of the vasculature, most specifically the  
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1 heart, but also dealing with wherever blood flows in  
2 the body.  
3 Q. And then, doctor, you received board  
4 certification in internal medicine in August of 1985;  
5 is that correct?  
6 A. Yes, ma'am.  
7 Q. And then you received a subspecialty board  
8 certification in cardiovascular medicine in November  
9 of 1989; is that correct?  
10 A. Yes, ma'am.  
11 Q. Now where do you currently practice cardiology?  
12 A. I am a consultant in cardiology at Minneapolis  
13 Cardiology Associates, at the Minneapolis Heart  
14 Institute, practicing primarily quaternary practice  
15 and tertiary practice out of Abbott Northwestern  
16 Hospital.  
17 Q. Could you take a moment to describe the work of  
18 the Minneapolis Heart Institute.  
19 A. The Minneapolis Heart Institute is a confederate  
20 of over now 50 cardiovascular specialists, surgeons,  
21 three surgical groups, one cardiology group, which is  
22 our group, which is approximately 29 cardiologists,  
23 cardiovascular anesthesiologists, interventional  
24 radiologists who deal mostly with peripheral vascular  
25 disease, and the pediatric cardiologists.

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1 Q. Is the Minneapolis Heart Institute the largest  
2 provider of cardiac care in the entire Twin City  
3 area?  
4 A. Yes, ma'am, it is.  
5 Q. And have you over time received national  
6 recognition for your work with the heart?  
7 A. The Minneapolis Heart Institute has  
8 approximately a 20-year history of being a premier  
9 single-specialty cardiovascular group. Dr. Robert  
10 Van Tassel, who founded the group, brought forth the  
11 concept of working with primary care physicians in a  
12 single-specialty group, giving high clinical  
13 medicine, but then returning the patient almost to  
14 the primary care physician for continued ongoing  
15 primary care.  
16 Q. Could you briefly describe for us the number of  
17 cardiovascular procedures that the Minneapolis Heart  
18 Institute does, say, in one year's time.  
19 A. We have roughly somewhere over 25,000 patient  
20 visits per year. We physically go to 28 outreach  
21 sites in Minnesota and western Wisconsin where one of  
22 our cardiologists will drive to Grand Rapids or  
23 Willmar or New Ulm to provide outreach consultative  
24 services to communities.

25 We perform about 4,500 diagnostic coronary  
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1 angiograms a year, roughly 1400 angioplasties,  
2 catheter-based interventions which we'll talk a  
3 little bit about a little bit later, about the same  
4 number of open-heart procedures a year, roughly 1400,  
5 putting in approximately 450 pacemakers last year,  
6 approximately 150 cardiac defibrillators. We have  
7 the active transplant program doing approximately 25  
8 heart transplants a year. So we try to offer the  
9 entire spectrum of clinical cardiovascular care.

10 Q. Now limiting your response now to your own  
11 practice, could you describe for the court and the  
12 jury what the nature of your practice is.

13 A. I am director of preventive cardiology at the  
14 Minneapolis Heart Institute. I spend approximately  
15 95 percent of my time in acute patient care. Of  
16 approximately -- of the 95 percent of my time, 75  
17 percent of the time is spent with the full spectrum  
18 of cardiovascular care, approximately 15 to 20  
19 percent is -- dealt with what's called primary  
20 prevention, which is trying to keep a patient from  
21 having the first heart attack, and I have a special  
22 interest in what we call secondary prevention, which  
23 is once a patient's had a heart attack, keep that  
24 patient from coming back again and again by  
25 addressing the causative agents that caused them to

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1 come for the first time.

2 Q. Doctor, are you also the chief operating officer  
3 and board chair of a company called ProMedicus  
4 Systems, Inc.?

5 A. I am.

6 Q. And could you briefly describe for us what the  
7 work of that company is?

8 A. It's an offshoot of our medical environment in  
9 the metropolitan area, which for most of you in this  
10 room I don't have to expound on. We saw the biggest  
11 issue over the past half a dozen years or so of being  
12 appropriate care, getting the right care to the right  
13 patient in the right setting, whether that's in a  
14 primary care setting or whether the patient needs to  
15 get to a specialist to try and maximize the  
16 efficiencies of serving that patient, so that  
17 wherever they come, they are served appropriately.

18 What we have done first through the Heart  
19 Institute and now through this company is to try,  
20 with a web-based computerized service, to give  
21 specialized help to primary care physicians so that  
22 they will have specialty help wherever a patient  
23 presents. The spectrum of medical knowledge is  
24 tremendous and the pressures on primary physicians  
25 are tremendous in this atmosphere with that

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1 environment that we live in, so we try and give as  
2 much help in -- in -- in line with what the mission

3 of the Heart Institute has been since day one, to  
4 work with primary care physicians to give best  
5 patient care wherever the patient presents.  
6 Q. Doctor, in the early '80s were you medical  
7 director and chief of staff at the McNamara Hospital  
8 and Nursing Home in Fairplay, Colorado?  
9 A. I was.  
10 Q. Please tell us your experience in working with  
11 that nursing home.  
12 A. Between my first and second year of residency, I  
13 took a one-year sabbatical, and with that ran a small  
14 hospital, 16-bed nursing home, emergency room and  
15 clinic that was 88 miles southwest of Denver. It was  
16 probably one of the best learning experiences I've  
17 ever had to understand what a primary care physician  
18 feels, especially in a rural area, when they don't  
19 have some specialist right around the corner.  
20 Q. And then between your residency and cardiology  
21 fellowship, did you work as director of utilization  
22 review for Midway Hospital here in St. Paul?  
23 A. I did.  
24 Q. And what was the nature of that work, doctor?  
25 A. Again focusing on quality of care, I was

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1 employed to review charts in order to try and make  
2 sure and work with physicians to make sure that  
3 patients were appropriately treated in a hospitalized  
4 setting.  
5 Q. Now doctor, are you a member of a number of  
6 medical associations and societies?  
7 A. I am.  
8 Q. Are you a member of the American College of  
9 Physicians?  
10 A. Yes, ma'am.  
11 Q. And the American Medical Association?  
12 A. Yes, ma'am.  
13 Q. And the American Heart Association and its  
14 Council on Clinical Cardiology?  
15 A. Yes, ma'am.  
16 Q. And have you served on the board of directors of  
17 the American Heart Association, Minnesota affiliate?  
18 A. Yes, ma'am.  
19 Q. And have you worked as the chair of the  
20 Physician Cholesterol Task Force of the American  
21 Heart Association, Minnesota affiliate?  
22 A. Yes, ma'am.  
23 Q. Is it fair to say, Dr. Graham, that some of your  
24 primary interests in cardiology are the secondary  
25 prevention of cardiac events, and the delivery of

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1 appropriate care throughout health care?  
2 A. Yes, ma'am.  
3 Q. Now that we've learned about your training in  
4 clinical cardiology, let me ask you these questions:  
5 Are you an expert in epidemiology?  
6 A. I am not.  
7 Q. Are you an expert in biostatistics or health

8 economics?

9 A. No, ma'am.

10 Q. Doctor, I'd like to begin our discussion today  
11 with the anatomy of the heart. We have heard from  
12 previous experts in this case about the gas exchange  
13 that takes place in the lung. I'd appreciate it,  
14 with the permission of the court, if you would step  
15 down and describe for us how the oxygenated blood  
16 gets from the lungs to the heart.

17 A. I first have to apologize for my drawing before  
18 I take the first stroke, but what we would like to  
19 show -- this is a heart, and the heart is a pump.  
20 And if you think of it very simply as a pump that you  
21 would pump out your basement with or anything else,  
22 it becomes an easier concept. The blood, after it is  
23 used up -- the blood basically delivers nutrients and  
24 oxygen. When the blood delivers its oxygen to the  
25 tissues it becomes blue, and we look at that as a

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1 darker color and we call that blue blood. And as the  
2 blue blood comes back to the heart from the top, the  
3 superior vena cava and the inferior vena cava, it  
4 comes back to a place called the right atrium. We'll  
5 just say RA there. And that right atrium then is  
6 filled with blue blood that goes into the right  
7 ventricle. Ventricles are the bottom chambers of the  
8 heart, atria are the top chambers. And the job of  
9 the right ventricle is to push the blood in a fairly  
10 low-pressure system to the lungs.

11 Now once it goes to the lungs, as Dr. Davies has  
12 very exquisitely talked about, it picks up oxygen and  
13 comes back to what is now the left atrium, the top  
14 chamber on the left side of the heart -- this is if I  
15 was facing you like this -- and then into the left  
16 ventricle, which is the biggest, most powerful  
17 chamber of the heart, and is responsible for pumping  
18 blood to the body. And the blood pressure that we  
19 take and that you may be familiar with, 120, 140,  
20 whatever like that, is generated in this pumping  
21 chamber, and then the red blood is directed to the  
22 body, all right, through the aortic valve out to the  
23 body. Okay? And once that happens, if this pump  
24 does not work appropriately, there is decreased flow,  
25 just as if the sump pump in your basement didn't

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1 work, you would have water in your basement. With  
2 that there is decreased flow, and there can be back-  
3 pressure of blood back to the lungs, which causes the  
4 person to be short of breath.

5 One of the things that Dr. Davies talked about  
6 is shortness of breath. There's also a question: Is  
7 it the heart or is it the lungs? Many of the people  
8 that I see it's the heart, where the heart is no  
9 longer pumping well, and the blood pushes back  
10 towards the lungs and makes somebody feel short of  
11 breath.

12 MS. NELSON: Dr. Graham, let's just mark

13 your picture as Trial Exhibit 25023. And Your Honor,  
14 at this time we would offer 25023 for demonstrative  
15 purposes only.

16 MR. MARTIN: No objection.

17 THE COURT: Court will receive 25023.

18 BY MS. NELSON:

19 Q. Now doctor, if you would take a look at Exhibit  
20 30111, is that, although enlarged, an anatomically  
21 correct model of the heart?

22 A. I believe it is.

23 MS. NELSON: Your Honor, we would at this  
24 time offer Exhibit 30111 for illustrative purposes  
25 only.

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1 MR. MARTIN: No objection.

2 THE COURT: Court will receive 30111.

3 BY MS. NELSON:

4 Q. Doctor, can --

5 Using the heart, can you explain to the jury how  
6 the body gets its blood supply?

7 A. I can. This again --

8 Your heart is about a little bigger than your  
9 fist, so if everybody makes a fist in this courtroom,  
10 if you have a big -- big fist, you probably have a  
11 big heart, and if you have a little fist, your heart  
12 is probably a little bit smaller, so this is many  
13 times bigger than any of our hearts. The arteries  
14 that flow on the surface of the heart are about as  
15 big as a lead pencil. Okay? And so they're --  
16 they're just a few millimeters in diameter.

17 And I want to just first open up this heart, and  
18 of course we can't do that in real life, but this is  
19 the right ventricle that pushes the blood out to the  
20 lungs, the blue blood, then it comes back to the  
21 heart, to the left atrium, this very thick muscle,  
22 the pumping chamber of the heart called the left  
23 ventricle. And if this doesn't work, if the pump  
24 doesn't work, then nothing works and you die.

25 And with that, when it's pumped out here to the

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1 aortic valve, it comes through what's called the  
2 aortic arch. These are the vessels that feed the  
3 head. The head is the most sensitive organ in the  
4 body, the brain -- six seconds it can only be  
5 deprived of oxygen and then it stops working. And  
6 then this aortic traverses down and feeds the various  
7 organs of the body there. And this is the pump, and  
8 I'm a little prejudiced being a cardiologist, but I  
9 think this is -- you know, if the heart doesn't work  
10 well, nothing else works well.

11 Q. Doctor, then how does the heart muscle itself  
12 get its blood supply?

13 A. The heart is like the hands, the legs, anything,  
14 it needs -- it needs blood and it needs oxygen, and  
15 there are various ways that the heart can get oxygen,  
16 but the -- the primary way and the way that you and I  
17 at this moment are getting our oxygen, I will sketch

18 out again for you, and I again --  
19 The aorta, as I -- as you saw it there, comes  
20 out and gives its branches to the head and to the  
21 arms and then continues on down like this. The  
22 arteries that feed the heart are run on the surface  
23 of the heart. And I will try my best to sketch those  
24 arteries out for you. And what they look like when  
25 we look at pictures of them is essentially tree roots

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1 that keep branching. And when they branch, they give  
2 the various nutrients, oxygen and sugar -- that's all  
3 that's delivered to the heart -- but it's crucial.  
4 The heart muscle like any other muscle needs the  
5 nutrients.

6 This again, if I was facing you, this would be  
7 the right coronary artery here -- and we will talk  
8 more about this as we go on -- this is called the  
9 left main coronary artery, because it's the main  
10 artery going in the left side of the heart, this is  
11 the left anterior descending coronary artery or LAD.  
12 In most people that's the most important artery  
13 because -- and I'll show -- because it feeds this  
14 thick front wall of the heart. This is the LAD  
15 coming down here on the surface of the heart.  
16 Convenient for bypass surgeons and all that it  
17 happens to be located on the surface. But with that,  
18 that's LAD comes down here, the circumflex coronary  
19 artery circles around the back of the heart. Okay?  
20 And that's usually a little smaller in most people,  
21 but you can see it circling around back here. All of  
22 us have these, that's how our heart get its blood  
23 supply. The blood supply then goes through the heart  
24 from the outside to the inside after these arteries  
25 continue to branch.

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1 MS. NELSON: Your Honor, we will mark Dr.  
2 Graham's drawing of the coronary arteries as Trial  
3 Exhibit 25024, and offer it for illustrative purposes  
4 only.

5 MR. MARTIN: No objection, Your Honor.

6 THE COURT: Court will receive 25024.

7 BY MS. NELSON:

8 Q. Now Dr. Graham, I'm going to ask you to get your  
9 notebook of slides, please.

10 If you would turn to Trial Exhibit 30018,  
11 please. Dr. Graham, is that an anatomically correct  
12 illustration of the arterial system?

13 A. Should I show them?

14 Q. No, not yet. Just "yes" or "no."

15 A. Yes.

16 MS. NELSON: Your Honor, we offer Exhibit  
17 30018 for illustrative purposes only.

18 MR. MARTIN: No objection, Your Honor.

19 THE COURT: Court will receive 30018.

20 BY MS. NELSON:

21 Q. If you would, Dr. Graham, please describe what  
22 is depicted on 30018.

23 A. Of course, again, a little biased cardiologist,  
24 but the heart is the center pumping organ. When the  
25 aortic arch comes around as I drew here, in much  
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1 nicer fashion is shown the innominate system here  
2 that gives a branch to the arm, the right carotid  
3 artery that feeds the right side of the brain, the  
4 left carotid system and the left subclavian, and then  
5 the aorta that comes down here and feeds the various  
6 intestinal organs, kidneys coming off here, and then  
7 this upside down Y is the iliac arteries that goes to  
8 the femorals and down and feeds the legs. Again it's  
9 much the same as the diagram that I show here, it's a  
10 branching tree almost like tree roots that then feed  
11 each of the respective organs downstream.

12 Q. Now doctor, I'd like to turn your attention to  
13 Trial Exhibit 30021 in your notebook, doctor.

14 A. Excuse me. Which number?

15 Q. 30021.

16 Is that an anatomically correct illustration of  
17 the coronary arteries that would aid you in  
18 illustrating your testimony?

19 A. Yes, ma'am.

20 MS. NELSON: Your Honor, we would offer  
21 30021 for illustrative purposes only.

22 MR. MARTIN: No objection, Your Honor.

23 THE COURT: Court will receive 30021.

24 BY MS. NELSON:

25 Q. Dr. Graham, would you please describe what we

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1 see on Exhibit 30021.

2 A. Again this is a much prettier drawing of the --  
3 of my drawing right here. And the aorta is coming  
4 out here. The valve that I show, the aortic valve,  
5 which is the trap door between the heart and the  
6 aorta, is under the pulmonary artery here, but you  
7 can see the right coronary artery coming out here  
8 again, branch -- giving branches off, the aorta  
9 coming out here. The very important big left  
10 anterior descending coming down the front of the  
11 heart where the big pumping portion is, and then the  
12 circumflex then circling around the back of the  
13 heart.

14 Q. Doctor, what is the size of a coronary artery?

15 A. As I had mentioned before, a coronary artery in  
16 most people is roughly the size of a pencil. And so  
17 when you -- when we talk about working inside that  
18 and doing angioplasties and stuff, we're basically  
19 working inside a pencil.

20 Q. Now doctor, I want to turn your attention to the  
21 subject of arteries developing blockages.

22 Could you briefly describe for us -- and you  
23 might as well stay right there because we're going to  
24 use exhibits here -- how arteries develop blockages.

25 A. Again, I -- I think a simple drawing can -- can

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1 tell a lot in something like this.

2 An artery is a pipe. It has to deliver the  
3 goods downstream like any of the pipes we have in our  
4 own home. I'm just going to draw for you what would  
5 be a pipe here. And with that we'll look end-on at  
6 the same pipe here. And over time an artery can  
7 develop -- and I might -- must admit by the  
8 time -- we're going to look at some angiograms later  
9 and all -- by the time we see something on an  
10 angiogram, it's a quite large blockage. And if you  
11 looked in all of our arteries by virtue of living in  
12 Western society, we would have abnormal arteries.  
13 And I'll explain a little bit more about that later.  
14 But by virtue of having higher lipids than is  
15 necessary, with other insults to the artery, over a  
16 period of time a small plaque can form in that  
17 artery. And it's just like any other blockage in a  
18 pipe or anything, and over time that can increase in  
19 size with that.

20 If we look then end-on, a beginning of flow  
21 limitation happens when that plaque blocks off that  
22 artery. We think most of the time a flow limitation  
23 has to be 70 percent or more to really impede blood  
24 flow down the artery.

25 Q. Now doctor, would you take a moment in your

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1 notebook and look at the following exhibits for me:

2 30022, 30040, 30015 --

3 A. Wait, wait, wait.

4 Q. Actually, 30015 is a model. 30033 and 30043.

5 Doctor, are each of these exhibits either actual  
6 microscopic photographs of coronary arteries or  
7 accurate portrayals of coronary arteries?

8 A. Yes, they are.

9 MS. NELSON: We would offer, Your Honor,  
10 30022, 30040, 30015, 30033 and 30043 for illustrative  
11 purposes only.

12 MR. MARTIN: No objection, Your Honor.

13 THE COURT: The court will receive those  
14 into evidence.

15 BY MS. NELSON:

16 Q. Turning your attention, Dr. Graham, first, to  
17 30022, could you please describe what this exhibit  
18 portrays.

19 A. What this exhibit portrays are two examples of a  
20 relatively normal artery here, again a pipe in  
21 cross-section here that looks relatively normal, and  
22 this would be a far advanced atherosclerotic plaque,  
23 "atherosclerosis" meaning a blockage that develops  
24 over time that has end-on narrowed the artery  
25 severely, on lengthwise, caused a -- probably we

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1 would call that an 80 percent or more blockage,  
2 impeding flow, blood flow in the artery.

3 Again with a microscope, though, when you look

4 at a coronary artery disease or atherosclerosis in  
5 general, it tends to be a diffuse disease, but with a  
6 focal clinical presentation. And we'll talk a little  
7 bit about that.

8 Q. Doctor, atherosclerosis, is that a disease of  
9 the artery that's characterized by the blockages that  
10 you've been describing?

11 A. Yes. Atherosclerosis, again, is a diffuse  
12 disease. And I think we will have to draw the  
13 distinction between atherosclerosis -- and again,  
14 almost everybody in this room, by virtue of living in  
15 Western society, has some degree of atherosclerosis.  
16 It's the question of who is going to present with  
17 clinical events, when is that atherosclerosis going  
18 to accelerate into a clinical event that becomes the  
19 most important thing.

20 Q. Now what risk occurs when the coronary artery  
21 creates blockages?

22 A. Again we talk of the coronary artery, if we look  
23 at the model or model there or something, if the  
24 blood doesn't flow down the pipe, within four to six  
25 hours all the muscles downstream die. And that's

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1 what heart attack is. A heart attack is when the  
2 area here closes off and a clot forms there, if  
3 the -- if -- the heart muscle downstream here then  
4 would die from that. And that's what a heart attack  
5 is.

6 Now when people come to the emergency room in  
7 most places in the country, they will be given a  
8 clot-dissolving medicine to try and reopen the  
9 artery. About 70 percent of the time it works, 30  
10 percent of the time, unfortunately, it doesn't. And  
11 only about 40 percent of patients who are eligible  
12 for that get to the emergency room in time to get the  
13 clot-dissolving medicine.

14 Q. Now focusing for a moment what is portrayed on  
15 that exhibit as -- it looks like little white lines,  
16 what does that depict?

17 A. What -- what the -- what the white lines here  
18 would depict, there is a lipid core here that -- that  
19 is -- and then there's a fibrous cap over that, the  
20 body caps that, and then that cap, for reasons that  
21 we really don't understand, can break down. When  
22 that happens, the goo of the lipid core, which is a  
23 little -- a little bit like a little Jell-O in all of  
24 our coronary arteries, can then pour out and it  
25 causes a clot to form there.

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1 Q. Now you used the term "plaque." What did you  
2 mean by that term?

3 A. "Plaque" just means blockage, I think. And I  
4 think in keeping it as simple as we can, which we try  
5 to do for patients to understand, is if a plaque  
6 remains stable, it can -- it can be there for a long  
7 time. But a plaque we say is basically a blockage.

8 Q. Now what is portrayed by the central gray area

9 in that coronary artery?  
10 A. This is a depiction, an artist's depiction of a  
11 clot that is formed after this area in the artery has  
12 cracked open and some of this goo has come out here  
13 and causing a blood clot to form right there.

14 Q. Now I would phoning us your attention on Trial  
15 Exhibit 30040 and ask you to describe what we see in  
16 that exhibit.

17 A. This is an actual photomicrograph of a coronary  
18 artery plaque. Again the coronary artery itself used  
19 to be this big, and over time a fat-filled lipid  
20 plaque has built up here. This is the fibrous cap,  
21 which is like a band that covers that, that is the  
22 border between where the plaque or the blockage is  
23 and where the blood flows down now. This area here  
24 has been filled with a white contrast material that  
25 opens the artery up, and this is a post-mortem

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1 examination.

2 This now we may consider to be a 60 percent, 70  
3 percent blockage. It is my job as a cardiologist,  
4 it's very important to find out whether that's in the  
5 left main coronary artery or whether it's way  
6 downstream here in the distal mainstream artery,  
7 because the treatment is much, much different. Any  
8 one can cause pain.

9 This is the type of patient who presents with  
10 what we call stable angina, and as the patient walks  
11 down the street, gets a little chest pain, stops and  
12 the chest pain goes away, because as the heart  
13 demands more blood flow, there is this blockage that  
14 has impeded the blood flow from getting there. And  
15 with that, we then have to work it up and say where  
16 is this blockage and what do we need to do about it?

17 People who have coronary blockage, as far as  
18 treating them, there's only three initial treatments;  
19 one is medicine, the second is angioplasty or  
20 stenting, the third is coronary bypass surgery for  
21 people who have severe or life-threatening disease.

22 Q. Now doctor, would you look at the model that we  
23 have that's been marked 30015 --

24 No, it's right here.

25 A. Oh, sorry.

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1 Q. Is that an accurate or inaccurate depiction of  
2 the clogging of a coronary artery?

3 A. This is an inaccurate depiction --

4 Q. Could you --

5 A. -- for most coronary arteries.

6 Q. Could you explain why.

7 A. Intuitively we used to think that you would have  
8 no blockage, then a 25 percent blockage, then a 50  
9 percent blockage, then it goes 60, 70, 80, 90, and  
10 finally to a hundred percent blockage. We've learned  
11 that that is not true. If you had to pick one  
12 blockage here that is most likely to cause a heart  
13 attack, this one; it's the 30 to 50 percent blockage

14 that suddenly, like a popcorn seed, pops, and when it  
15 pops, it can pop like one of Orville Redenbacher's  
16 finest and go out into the lumen of the artery and  
17 block it off, or it can pop very small and you never  
18 know it happened.

19 We know that the primary cause of acute heart  
20 attacks and unstable coronary syndromes, which means  
21 unstable angina, are -- is this disruption of the  
22 plaque and a clot forming there. So we know that  
23 there are certain conditions that people's blood is  
24 stickier. When they do rupture this plaque, that  
25 they form -- are more likely to form a clot. The

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1 primary one and the most easily reversible one is  
2 cigarette smoking. Because when the blood goes  
3 through the lungs, as Dr. Davies eloquently defined  
4 to you, it goes single file. Those red cells and the  
5 platelets, which are responsible for blood clotting,  
6 when exposed to cigarette smoke, the platelets become  
7 angry or turned on, and so just as when you skin your  
8 knee, you may form a very good scab or clot there,  
9 when your artery has a skinned knee, you have a hyper  
10 response to that and form of clot that can close the  
11 artery and cause an acute heart attack.

12 Q. Dr. Graham, let's go back a minute. You used  
13 the word "angina." Could you explain what the word  
14 "angina" means?

15 A. Angina is chest pain, and it's usually caused by  
16 low blood flow to the heart. So any time somebody  
17 has -- we call it stable angina, and that would be a  
18 plaque like this where they have exertional angina  
19 and then they rest and it goes away, and we juxtapose  
20 that to unstabilized angina where they have resting  
21 pain or they've had a change in the pattern of their  
22 angina.

23 Q. Okay. Let's take a look at Trial Exhibit 30033.  
24 Does this exhibit portray the rupture of the plaque  
25 that you just described in the unstable angina?

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1 A. I think this is a beautiful depiction of what  
2 happens in patients who have either unstable angina  
3 or a heart attack. This maybe just a few hours ago  
4 was a 50 percent blockage here with a fibrous cap  
5 here covering this. At that time, then, that popcorn  
6 seed popped, if you would, and the artery here -- the  
7 goo came out here, you get intense vasospasm here,  
8 this -- this relatively normal artery contracts down,  
9 formed a clot that was a fatal event in this person.

10 And we know now from numerous studies and from  
11 treating hundreds and thousands of patients like  
12 this, the patient who was alive that came in with a  
13 heart attack, by knowing this, we give them a  
14 clot-dissolving medicine, called TPA, in the  
15 emergency room, and again in about 70 percent of  
16 patients it opens up the artery within an hour and a  
17 half.

18 Q. Turning your attention then to Trial Exhibit

19 30043, does this exhibit, doctor, portray a clot in  
20 an artery that has atherosclerosis?  
21 A. This again is a depiction now in a length-wise  
22 form, this has to be a pipe, again, that has to  
23 deliver blood to the muscle downstream of a -- what  
24 was a stable plaque that has now fissured or ruptured  
25 in a length-wise fashion now. You can see different

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1 colors of clots there. We know that this is coming  
2 and going, and then the fatal event in this patient  
3 was a big red cell cast here that closed off the  
4 artery entirely.

5 We know that 30 percent of patients who have  
6 heart attacks never make it to the front door of the  
7 hospital, and what happens is when they occlude the  
8 artery, they either drop their blood pressure or have  
9 a lethal arrhythmia.

10 Q. Doctor, I'd ask you to look in your book at  
11 Trial Exhibit 30020.

12 A. 30020.

13 Q. Is that an anatomically correct depiction of the  
14 aftermath of a heart attack?

15 A. Yes, it is.

16 MS. NELSON: Your Honor, we would offer  
17 30020 for illustrative purposes only.

18 MR. MARTIN: No objection, Your Honor.

19 THE COURT: Court will receive 30020.

20 BY MS. NELSON:

21 Q. Could you explain 30020 for the jury, doctor,  
22 keeping in mind the previous slide which demonstrated  
23 the fatal clot.

24 A. If you think again of a blockage, and a heart  
25 attack is a total closure of the artery, if the

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1 artery is not opened within four to six hours, the  
2 muscle downstream again, fed by this end of the root,  
3 if you would, dies, and that is now scar tissue  
4 there. And the pumping function that the heart  
5 needed -- needs to do, and this would correlate on  
6 this model to the tissue here and the tissue on the  
7 end of the heart, is now gone, and that area becomes  
8 not thick, but thin, and no longer pumps. So the  
9 patient is now compromised to the point where they  
10 have heart muscle that doesn't work. And if it's a  
11 sufficient amount of heart muscle, they can go into  
12 what we call congestive heart failure. Again, the  
13 muscle -- there is a certain amount of muscle needed  
14 to pump, and if you lose that amount of muscle  
15 needed, the blood backs up into the lungs, you get  
16 short of breath, and you don't pump enough blood  
17 forward, and the vital organs of the body are not  
18 perfused or they're not getting enough blood.

19 The other point about this also, this area which  
20 was now dead conducts electricity differently, and  
21 people are at risk from dying from what we call  
22 arrhythmias, or the heart beating chaotically after  
23 they have a full-thickness scar in their heart after

24 a heart attack.  
25 Q. Doctor, I want to turn your attention now to the  
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1 impact smoking has on this disease of the blockage of  
2 the coronary arteries that we've been talking about.

3 Doctor, based on your training and experience,  
4 do you have an opinion to a reasonable degree of  
5 medical certainty as to the mechanisms by which  
6 smoking are a cause of these blockages in the  
7 arteries?

8 A. I think, again, for everybody in this room's  
9 benefit, I think that it's important to realize that,  
10 you know, there's a lot of vascular disease in this  
11 country and that we have to do everything we can to  
12 keep, you know, all of us out of having vascular  
13 disease.

14 There are four prime mechanisms which smoking  
15 causes not only the development of atherosclerosis,  
16 but the development of what we call acute events.

17 MR. MARTIN: Your Honor, I'm going to  
18 object to this. He has not responding to the  
19 question that was asked. I request that he do.

20 THE COURT: I think you should re-ask the  
21 question, counsel.

22 MS. NELSON: That's fine.

23 BY MS. NELSON:

24 Q. Doctor, just tell me now, do you have an  
25 opinion, "yes" or "no," to a reasonable degree of

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1 medical certainty, as to the mechanisms by which  
2 smoking is a cause of this disease of the blockage of  
3 the arteries?

4 A. Yes, I do, ma'am.

5 Q. And what is your opinion, doctor?

6 A. Again, I believe that there are four main ways  
7 which, looking at the model, that we have established  
8 that smoking is known to be deleterious to coronary  
9 arteries. The first is that it competes for oxygen.  
10 As I said, the blood flowing to the heart has to  
11 deliver sugar and oxygen. That's what it does. The  
12 heart doesn't use fat. And in people with lung  
13 disease, as Dr. Davies has again eloquently said, you  
14 don't get as much oxygen into the blood, and so if a  
15 normal amount of saturation of oxygen is a hundred,  
16 some -- some people may have saturations at 80, 85,  
17 you've decreased the amount of oxygen in the blood.

18 The second way it competes for oxygen is there  
19 is carbon monoxide, CO poisoning in cigarette smoke,  
20 and most smokers have about ten percent of their  
21 hemoglobin, which is responsible for carrying oxygen,  
22 that's what hemoglobin does, carrying oxygen in the  
23 blood to the various body tissues, ten percent of the  
24 hemoglobin is tied up as carboxylated hemoglobin, or  
25 is tied up in this almost irreversible form.

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1 Q. Doctor, is hemoglobin commonly known as the red  
2 blood cells?

3 A. The red blood cells are -- contain hemoglobin.  
4 And hemoglobin's job, again it's only job is to carry  
5 oxygen to the tissues and deposit it in the tissues.  
6 So we have a double-fold, there's a competition for  
7 oxygen because of lung damage that we can't get the  
8 oxygen into the blood, secondly, the hemoglobin is  
9 then tied up, about 10 percent of it, for -- with the  
10 carbon monoxide.

11 Q. Doctor, if you'd look in your book at Trial  
12 Exhibit 30045, please. Does that picture accurately  
13 portray the concept of carbon monoxide competition  
14 for oxygen?

15 A. Yes.

16 MS. NELSON: We would offer Trial Exhibit  
17 30045, Your Honor, for illustrative purposes only.

18 MR. MARTIN: No objection, Your Honor.

19 THE COURT: Court will receive 30045.

20 BY MS. NELSON:

21 Q. Now could you describe for the jury and the  
22 court what you see in that exhibit, doctor.

23 A. This is the alveoli of the -- where air exchange  
24 happens in the -- in the lung. And it's analogy to a  
25 honeycomb with a lot of --

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1 And this layer here is only one cell thick, so  
2 as the red cells come through and the platelets, they  
3 are exposed to, as the ingredients, oxygen, but also  
4 carbon monoxide diffuses through there and ties up  
5 the red cells so it cannot accept the oxygen  
6 molecule. We also know that from previous -- I'm  
7 sure Dr. Davies talked about that there -- as this is  
8 broken down, what I talked about, competition for  
9 oxygen, if this -- you lose some of the architecture  
10 here, the ins and outs, it's harder to get anything  
11 across here into the bloodstream.

12 Q. Now doctor, you mentioned that there were four  
13 mechanisms by which smoking caused these blockages in  
14 the artery. What is the second mechanism?

15 A. The second mechanism, and I will explain what  
16 this means, is what we call vaso -- "vaso" meaning  
17 vessel -- constriction. Vasoconstriction means a  
18 clamping down of a vessel. Okay. And what I will --

19 An artery, under the effects -- even a normal  
20 artery -- of cigarette smoke that is this big, then  
21 becomes this big under the direct toxic effects.  
22 There's smooth muscle around the artery that causes  
23 it to contract, and all of our -- all of our heart  
24 arteries have muscle around them, and it causes the  
25 artery to become smaller.

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1 It becomes very important if an artery has, say,  
2 a 50 percent blockage, and then you have  
3 vasoconstriction, the blockage will remain the same  
4 size here to here, what suffers is the lumen, and

5 suddenly what was a 50 percent blockage can become a  
6 70 to 80 percent blockage and cause restriction of  
7 blood flow.

8 Q. Doctor, would you look at Trial Exhibit 30039.  
9 Does that accurately depict the impact of smoking by  
10 vasoconstriction on the arteries?

11 A. Yes, ma'am.

12 MS. NELSON: We would offer, Your Honor,  
13 Trial Exhibit 30039 for illustrative purposes only.

14 MR. MARTIN: No objection, Your Honor.

15 THE COURT: Court will receive 30039.

16 BY MS. NELSON:

17 Q. Could you please demonstrate to the jury the  
18 concept of vasoconstriction caused by smoking,  
19 doctor?

20 A. Yes, ma'am.

21 Again in a much prettier way than I've shown you  
22 hear, an artery that is this big under the effects of  
23 cigarette smoke becomes constricted down, and  
24 therefore the flow of blood down there is limited  
25 along the entire length of it.

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1 Q. Doctor, what is the third mechanism by which  
2 smoking causes blockages in the coronary arteries?

3 A. We would best characterize this by a direct  
4 toxic effect.

5 Q. Now what do you mean by that?

6 A. In the arterial wall there is a buildup of  
7 cholesterol and there is an imperfect healing, if you  
8 would, of that plaque, that little bit of Jell-O in  
9 the arterial wall. Almost like sandpaper, the  
10 ingredients of cigarette smoke irritate that plaque  
11 when people are smoking, and it causes the lining of  
12 that to be a continual skinned knee, if you would,  
13 because there's a -- almost of a sandpaper going over  
14 that blockage up and down the artery from the direct  
15 effect of cigarette smoke.

16 Q. What is the fourth mechanism by which smoking  
17 causes blockage of the coronary artery?

18 A. The fourth mechanism, and -- and the one that --

19 When we talked about the first three, we were  
20 talking about the development of atherosclerosis, or  
21 the development of blockage. Again, many people in  
22 this room have atherosclerosis in various stages who  
23 have not been in the hospital or have not had bypass  
24 surgery or angioplasty for that.

25 The fourth event is probably the most important

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1 as far as clinical event, and that is what I would  
2 call angry or turned-on platelets.

3 Q. And what are platelets, doctor?

4 A. Platelets are parts of the blood that are  
5 responsible for blood clotting. And when you skin  
6 your knee and you begin to -- the blood stops there,  
7 and there's an initial mesh that forms over there to  
8 stop the bleeding, that is platelets that go to the  
9 area of injury. When you injure an artery or your

10 knee, there needs to be response to that. Again,  
11 through the lung, when the platelets go through in  
12 single file with the red cells through the lung and  
13 are exposed to cigarette smoke, they get what we call  
14 turned-on or activated platelets. They are ready to  
15 clot. So when somebody has the ruptured plaque  
16 within the coronary artery, and people oftentimes  
17 will rupture plaque and they may heal down, they may  
18 not, but when somebody is a smoker they have a --  
19 what we call hypercoagulable state or a high  
20 coagulation state, and with that they tend to form  
21 clots more easily, they tend to present with unfatal  
22 symptoms or infarctions more often when they rupture  
23 the plaque.

24 We know that aspirin as an anti-platelet  
25 agent -- and many people in this room may take one  
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1 aspirin a day -- greases platelets so they don't  
2 stick together. We know from studies that it  
3 decreases the incidence of heart attacks in  
4 middle-aged males by about 20 to 25 percent, one  
5 aspirin per day. But the effects of smoking can even  
6 overwhelm the effects of aspirin, and that's been  
7 well-documented in the literature.

8 Q. Now doctor, in addition to smoking, are there  
9 other independent causative agents which  
10 substantially contribute to coronary artery disease?

11 A. Yes.

12 Q. And what are those?

13 A. When we look at causative agents of coronary  
14 artery disease, there are what we call the big four,  
15 which are smoking, cholesterol problems, either --  
16 diabetes mylodus or high blood sugar, and high blood  
17 pressure. These are the big -- what we call the big  
18 four causative agents of -- of coronary artery  
19 disease.

20 Somebody who has one of them only may not  
21 present with coronary disease. Yet the more you have  
22 of these, there's a synergy between them that makes  
23 you more at risk for presenting with coronary  
24 disease. So we look for people with multiple risk  
25 factors, we look for people with family histories of

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1 problems with that, but at the same time, these are  
2 the major things that almost every clinician is  
3 trained to look and ask people to say are you at risk  
4 for having a heart attack.

5 Q. Dr. Graham, is smoking, however, an independent  
6 causative agent which substantially contributes to  
7 the onset of coronary artery disease?

8 MR. MARTIN: I'm going to object to the  
9 form of the question.

10 THE COURT: No, you may answer that.

11 THE WITNESS: Excuse me?

12 THE COURT: You may answer that.

13 THE WITNESS: Okay.

14 A. In -- in my clinical training and clinical

15 experience, I believe that smoking is the most  
16 powerful modifiable risk factor for coronary artery  
17 disease and causative agent.  
18 Q. Now Dr. Graham, can you think of an analogy  
19 which would explain to the jury and the court the  
20 effect of smoking synergistically with the rest of  
21 these causative risk factors?  
22 A. Excuse me one moment, please.  
23 Q. Sure.  
24 (Witness pours himself a glass of water.)  
25 A. As physicians, and especially in dealing with  
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1 people with coronary artery --  
2 MR. MARTIN: Excuse me, Your Honor. It  
3 seems appropriate, perhaps, that the doctor return to  
4 the witness chair.  
5 THE WITNESS: Do you want me to do that?  
6 MS. NELSON: That's fine.  
7 THE COURT: Are you finished using the  
8 board?  
9 MS. NELSON: Well there's a number of  
10 devices down here, but we can answer this question  
11 there, but -- but he will be returning down to  
12 explain some things.  
13 THE WITNESS: I'll get my book.  
14 THE COURT: I wonder if we should take a  
15 short recess at this time.  
16 MS. NELSON: Sure. That's fine, Your  
17 Honor.  
18 THE CLERK: Court stands in recess.  
19 (Recess taken.)  
20 THE CLERK: All rise. Court is again in  
21 session.  
22 (Jury enters the courtroom.)  
23 THE CLERK: Please be seated.  
24 BY MS. NELSON:  
25 Q. Dr. Graham, before the break, I believe I had  
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1 asked you if you can think of an analogy which  
2 explains to the jury the effect of smoking and these  
3 other causative agents on the blockages in the  
4 artery, in the coronary arteries that we've discussed  
5 in the past.  
6 A. I think it would be helpful to -- to think of a  
7 wheat field, and if there is wheat to be grown in  
8 that field, you need to think of cholesterol and  
9 sugar as -- as fertilizer for that wheat. And let's  
10 speak to cholesterol, since it has probably been  
11 talked about a lot, and a lot of us think about that.  
12 If there is no cholesterol, if there is no  
13 fertilizer at all, there's essentially no crop and  
14 there's no wheat to grow. And about three percent of  
15 the people in our population have very, very low  
16 cholesterols and -- and therefore probably don't have  
17 any risk of coronary artery disease. These are  
18 cholesterols less than 130. You know, we don't see  
19 them very often. We don't see those patients in our

20 catheterization laboratory.

21 The truth of the matter, then, is for the other  
22 97 percent of the population, about five percent of  
23 those people have just the right amount of  
24 cholesterol. And by virtue of living in Western  
25 society, most of us have too much cholesterol, and

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1 the wheat grows fast and furious and oftentimes, like  
2 a yard that doesn't get cut, overgrown with each  
3 other. And the same could be said for diabetes.

4 I liken hypertension to wind that can blow the  
5 crop, and even in severe cases lay it down, but when  
6 the wind goes away, the crop recovers.

7 I liken smoking to a vigorous, sudden summertime  
8 hail storm that comes in, and if there's a crop  
9 there, the -- the hail is -- damages the crop and  
10 will forever more -- while it's there, it continues  
11 to damage the crop, but even after the hail is gone  
12 or when somebody quits smoking, the crop is  
13 irreparably damaged forever and will never be the  
14 same as it would have been.

15 Q. Doctor, you spoke of a patient presenting  
16 with -- I think you used the term "stable angina" or  
17 "stable coronary symptoms." When that patient  
18 presents to you and complains of chest pains, are  
19 there non-invasive diagnostic tools available for you  
20 to evaluate the damage to their artery?

21 A. Certainly.

22 Q. And what are those?

23 A. When a patient comes, we -- we take a history,  
24 first of all, and -- and see what the patient's  
25 causative agents or risk factors are, and we'll ask

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1 if they smoke, if they have high cholesterol, if they  
2 have diabetes, hypertension, if they have a family  
3 history of coronary events, the usual questions to  
4 see how many risk factors there are, because we know  
5 the more risk factors the more likely is that  
6 patient's pre-test risk of having blockages in their  
7 coronary arteries. Then we ask the type of pain they  
8 have, whether it's the typical stable type pain where  
9 they walk down the street, they get chest pains, they  
10 stop, it goes away, and they start again, it comes.  
11 That's typical angina as opposed to somebody who may  
12 say I have a sharp pain in my left -- the right side  
13 that kind of comes and goes, that would be atypical  
14 for low blood flow to the heart.

15 And then we try and quantify the amount of  
16 angina. Do they get angina when they're running  
17 vigorously two blocks, or do they get angina when  
18 they walk across the room? And there's something  
19 called a Canadian angina classification between one  
20 and four, one being angina for chest pain with  
21 vigorous exercise, four being all the way down in a  
22 stepwise fashion to resting chest pain. And so we  
23 qualify a patient that way, and then if it is safe,  
24 we usually do a treadmill test if the patient is

25     stable, if they don't have resting chest pain, in  
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1 order to make sure that they can re-create their  
2 symptoms on the treadmill, and then with the  
3 electrocardiogram, which is the heart monitors that  
4 we put on them to look at their cardiographic  
5 tracings that show evidence of low blood flow to the  
6 heart.

19 Q. Based on your experience, doctor, what is the  
20 range of cost for that type of stress testing?  
21 A. A regular treadmill stress test with just a  
22 electrocardiogram and the belts, the walking-type  
23 treadmill that is a standardized fashion, costs about  
24 \$250.00. Adding different types of imaging agents to  
25 that, there are various expenses associated with it,

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1 increases the cost anywhere from 600 dollars upwards  
2 to 3.000 dollars.

6 A. Yes, ma'am.

9 MR. MARTIN: No objection, Your Honor.

11 BY MS. NELSON:

14 A. In this stylized drawing, there is a patient on  
15 a treadmill, much as like the treadmills that you  
16 would see at an exercise store, but the treadmill is  
17 a very standardized one that is linked to a computer,  
18 and the elevation and speed of the treadmill is -- is  
19 standardized, so that whether I do a stress test in  
20 Boston, in Minnesota or San Francisco, there is a  
21 standardization of the -- of the testing.

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1 electrocardiograms going across there, are monitored  
2 for evidence of low blood flow to the heart.  
3 Q. Now doctor, before you spoke of a patient in an  
4 emergency condition who appears or presents with  
5 unstable angina or unstable coronary symptoms, such  
6 as a ruptured plaque or heart attack, what steps do  
7 you take when that patient arrives at your hospital?  
8 A. Patients who have ruptured a plaque and who  
9 present with unstable symptoms range from very high  
10 concern to an acute medical emergency. When somebody  
11 presents and we think the artery is closed, we have  
12 four to six hours to open that artery in some fashion  
13 in order to re-establish blood flow down so the heart  
14 does not become starved. So we view the ruptured  
15 plaque and a history consistent with that as a  
16 medical emergency. The patient is hospitalized,  
17 he -- he or she is often given blood thinners  
18 intravenously. If we think they have an acute heart  
19 attack, in some hospitals they would be given a  
20 clot-dissolving medicine. At the Minneapolis Heart  
21 Institute, at Abbott Northwestern, we will take that  
22 patient directly to the cardiac catheterization  
23 laboratory and try and open the artery directly with  
24 a balloon or wire, because we have a 95 percent  
25 chance of opening it directly with some of the tools

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1 that I will show you in a few minutes, as opposed to  
2 about a 70 percent if we give the -- give the  
3 clot-dissolving medicine.  
4 Q. Okay. Doctor, let's talk for a moment, then,  
5 about cardiac catheterization.  
6 Can you explain to the jury what an angiogram  
7 is.

8 A. As opposed to the treadmill testing -- I suppose  
9 I should have said this, but as you see the picture  
10 up there, a routine treadmill in somebody who has  
11 coronary disease has about an 80 percent what we call  
12 sensitivity of identifying patients with disease, and  
13 specificity, which means that if it is abnormal,  
14 we're pretty sure the patient has disease. That  
15 means eight out of 10 patients are identified. More  
16 importantly that means two out of ten are not.

17 At the same time, if we add one of those imaging  
18 modalities to it, either stress echo or nuclear  
19 stress test, the sensitivity and specificity of the  
20 test goes up to about 90 percent, which means we find  
21 about nine out of 10 patients with chest pain.

22 The gold standard is for people that we think  
23 have a very abnormal stress test or who are unstable  
24 and we're worried about them going on to a heart  
25 attack, becomes a coronary angiogram where we

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1 actually numb up the groin area and put a catheter in  
2 the femoral artery, thread that up in reverse flow  
3 from where I showed you before coming down, with  
4 special catheters and engage both the left and right  
5 coronary arteries and inject dye down the coronary

6 arteries to say where are their blockages, how severe  
7 are they, and how should they be treated. If the  
8 patient has blockages, initially there's only three  
9 treatments: There is medication, either oral or  
10 intravenously; and there is an angioplasty and all  
11 the things we do like angioplasty, stents,  
12 Roto-Rooters, things like that, and for the patients  
13 who have severe or life-threatening diseases, there's  
14 coronary bypass surgery.

15 Q. Let's start then with angiogram, and if you  
16 would look at Trial Exhibit 30044, 30028, and 30038,  
17 do these --

18 A. Can you read --

19 Can you give the second one, please?

20 Q. Sure. 30028 and 30038.

21 Do these photographs and pictures accurately --  
22 accurately anatomically depict the process of a  
23 coronary angiogram?

24 A. Yes, ma'am.

25 MS. NELSON: We would offer, Your Honor,  
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1 30044, 30028, and 30038 for illustrative purposes  
2 only.

3 MR. MARTIN: No objection, Your Honor.

4 THE COURT: Court will receive 30044,  
5 30028, 30038.

6 BY MS. NELSON:

7 Q. Focusing your attention, doctor, then, on 30044,  
8 can you describe for the jury, first of all, who's  
9 the physician in the picture?

10 A. That's me.

11 Q. Okay. And what are you doing there, doctor?

12 A. In the catheterization laboratory where this is  
13 depicted, with a hat and gown, with an assistant,  
14 another assistant in the background, the Cat Lab  
15 costs about two million dollars. And this is the  
16 patient's right groin here, it has been anesthetized,  
17 and I'm holding in my hand a percutaneous needle  
18 which I will enter the artery with.

19 Q. And doctor, is this an entire procedure done  
20 while the patient is awake?

21 A. The patient is awake, but sedated.

22 Q. Okay. Turning your attention, then, to 30028,  
23 can you describe for us what you see here? And I'd  
24 ask you to step down at this point because we will be  
25 using some medical devices, with the permission of

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1 the court.

2 THE WITNESS: Is that okay?

3 Thank you.

4 A. What this depiction shows, and I think we should  
5 start over here first, we usually enter the right  
6 femoral artery. Again if you think of the diagram  
7 that was shown of the blood flowing this way to the  
8 leg, we enter the largest artery we can -- the right  
9 femoral artery is about as big as your little  
10 finger -- and that decreases our chance of

11 complications because we actually have to perc and  
12 enter -- percutaneously enter the artery. And we  
13 then put a sheath in and then backflow into the heart  
14 up here what's called the coronary catheter that  
15 injects dye down those coronary arteries, watching  
16 under a fluoroscope as we do in making a movie x-ray  
17 of the -- of the blockages in the artery.

18 Q. Dr. Graham, are what has been marked as Exhibit  
19 30007 -- over here, doctor -- 30008 and 30010, are  
20 those medical devices that you would routinely  
21 utilize in performing an angiogram?

22 A. They are.

23 MS. NELSON: Your Honor, we would offer  
24 30007, 30008 and 30010 for illustrative purposes  
25 only.

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1 MR. MARTIN: No objection, Your Honor.

2 THE COURT: Court will receive 30007,  
3 30008, 30010.

4 BY MS. NELSON:

5 Q. Now doctor, if you could actually demonstrate  
6 how you perform the angiogram for the jury.

7 A. Again, the patient is brought to the  
8 catheterization laboratory in a lightly sedated  
9 state, and with that they are -- a few minutes is  
10 taken to sterily prepare the patient with iodine  
11 solution, and they are covered with a sterile drape  
12 from their chin all the way covering their feet. We  
13 then take some novocaine and anesthetize locally the  
14 area that we are about to enter. Once we have  
15 done -- placed a needle in the artery -- and again it  
16 takes a fair amount of training, we just perc one  
17 side of the artery -- we put a wire through the  
18 needle and pull the needle out so all that is in the  
19 artery is a wire. At that time we then, over the  
20 wire, place what's called a sheath, and the sheath  
21 stays in for the duration of the -- of the procedure.  
22 And there's a dilator and it stretches the artery,  
23 you'll see it's kind of a smooth bullet tip, as it  
24 goes into the artery. We then pull out the dilator,  
25 and there's a ball valve here that keeps the blood

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1 from coming back. We then pull the blood back and  
2 flush sterile heparinized saline in there, a blood  
3 thinner saline so the blood does not clot, and then  
4 we do our procedure through this tube that now  
5 resides in the groin or in the right femoral artery.

6 To reach the coronary artery, once we have done  
7 that, there is -- we have coronary catheters, which  
8 are directed over a guidewire up the aorta, and once  
9 this is in place and sitting in the coronary  
10 artery -- excuse me, in the femoral artery, we then  
11 put in --

12 This is a right coronary catheter, and the  
13 catheters are preshaped. This is made by -- made by  
14 SciMed, which is a Minnesota company. And we direct  
15 the --

16           You'll notice there's a J on the end of it.  
17   This is called a J wire. And I told you before that  
18   even though you couldn't see blockages in arteries,  
19   that they -- they were there, and -- and often times  
20   people's aorta are a cobblestone of what we call  
21   atherosclerosis. So if we put up a bare catheter, we  
22   would drag it against that cobblestone and risk  
23   knocking off pieces that would go down to the legs  
24   and cause problems. So we always use a J wire, which  
25   presents a blunt tip as it goes up the aorta against

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1   the blood flow and into the first part of the aorta.

2           Again this is now shown diagrammatically here.  
3   When the catheter comes up here, this is a left  
4   coronary catheter that engages the artery.

5           So as we advance the catheter, the case -- the  
6   entire case is done through this little tube that has  
7   been placed previously.

8   Q. And after you've injected dye into the coronary  
9   arteries, what is it that you're able to see?

10   A. We're looking for blockages, and you -- we --

11           The camera swings around the patient, and --  
12   and -- and once we direct this up to the coronary  
13   artery, I turn it here to direct it into the artery,  
14   and then we inject dye down the artery and take  
15   pictures as the camera swings around.

16           MS. NELSON: Your Honor, could we have the  
17   bailiff or the clerk pass this around, or is it  
18   permissible to do it in this fashion?

19           THE COURT: You don't plan to use that  
20   again, doctor?

21           (Laughter.)

22           THE WITNESS: Not unless you're  
23   volunteering.

24           THE COURT: Then you can pass it around.

25   A. So that's the diagnostic portion of -- of the

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1   case. And again, if we find blockages in there,

2   there are -- there are only three treatments:

3   medicines, angioplasty or bypass surgery.

4   Angioplasty works best on people who have one or two  
5   vessels blocked. And if there are multiple or severe  
6   blockages, bypass surgery tends to work better.

7   Q. Now doctor --

8   A. So --

9   Q. Doctor, excuse me, but looking back up at the  
10   screen for a moment, up to the left-hand portion, it  
11   appears to be a portrayal of coronary arteries on a  
12   screen; is that correct?

13   A. Yes, ma'am.

14   Q. And is that what you see when you look at an  
15   angiogram?

16   A. Yes. You look under the scope and then you'll  
17   see -- and we get an initial picture on a videotape  
18   in the room, and then we develop regular  
19   35-millimeter film or digital film and look at them  
20   after in a more refined fashion. But we almost

21 always in the loom know what's going on while we're  
22 doing the case.  
23 Q. And Dr. Graham, what is the typical cost for one  
24 angiogram?  
25 A. The typical cost for one coronary angiogram is  
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1 6,000 dollars.  
2 Q. Okay. Doctor, now you've done the angiogram and  
3 discovered that your patient indeed has some  
4 blockages. I would like to turn our attention now to  
5 the interventions that you've mentioned to the jury  
6 several times available to attempt to fix the  
7 ruptured plaque or the clot. And let's start with  
8 coronary angioplasty.  
9 A. Okay.  
10 Q. Could you take that back from the jury. Thank  
11 you.  
12 A. Oh, sure. Excuse me.

13 (Physical exhibit handed to the witness.)

14 Q. Could you describe for the jury what coronary  
15 angioplasty is, without the device at this point.  
16 A. All right. Coronary angioplasty is through the  
17 same type of catheter that I showed you, if that was  
18 a right coronary lesion, we would thread a very small  
19 catheter and a wire with a balloon on the end of  
20 the -- of the catheter, and then again watching under  
21 the fluoroscope, we would first direct a wire across  
22 the blockage, and there's a skill to threading that  
23 very thin 14-one-hundredths-of-a-millimeter-thick  
24 wire across that -- that artery or the blockage, and  
25 then we direct the balloon across and inflate the

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1 balloon to stretch the artery at that point.  
2 At that point there is no blood flow down the  
3 artery and you need to put down the balloon and pull  
4 it out. And there's a skill that goes with that.  
5 Q. Doctor, I show you what's been marked as Trial  
6 Exhibit 30014. Is this an accurate portrayal of an  
7 angioplasty?  
8 A. Yes.  
9 MS. NELSON: Your Honor, we would offer  
10 30014 for illustrative purposes only.  
11 MR. MARTIN: No objection, Your Honor.  
12 THE COURT: Court will receive 30014.

13 BY MS. NELSON:

14 Q. Could you describe to the jury what appears on  
15 that exhibit.  
16 A. This in cross-section -- we only have half of  
17 our pipe here, appears to be a very significant  
18 blockage in highly stylized form. And again this --  
19 remember we're working inside a pencil, this has been  
20 blown up to a huge degree to show us. You put the  
21 balloon across, expand the balloon to push out the  
22 artery at that area. When you put down the balloon,  
23 you hope for a very good cosmetic effect to open up  
24 the artery, and a functional effect.  
25 Q. Now doctor, turning your attention to several of

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1 these medical devices that have been marked as 30003,  
2 30014 and 30009, are these medical devices that you  
3 would typically use to perform a coronary  
4 angioplasty?

5 A. Which ones?

6 Q. '3, '14 and '9.

7 A. Yes, they are.

8 MS. NELSON: Your Honor, we would offer  
9 30003, 30014 and 30009 for illustrative purposes  
10 only.

11 THE COURT: Court will receive 300013,  
12 300014, 300009.

13 MS. NELSON: Let me just say that again,  
14 Your Honor, I think I may have misread them.

15 THE COURT: Okay.

16 MS. NELSON: It's 30003, 30014, and 30009.

17 THE COURT: All right.

18 BY MS. NELSON:

19 Q. Dr. Graham, can you describe the angioplasty  
20 procedure again for the jury?

21 A. Again, we spent years doing cardiology  
22 fellowships to learn how to do this procedure, and I  
23 don't expect to -- to teach each of you about the  
24 procedure. But I would just advance this wire, which  
25 we placed through the angioplasty catheter, and then

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1 the end of this wire, you'll see, has a very small J  
2 tip on it, and we -- we dance that artery -- we dance  
3 that down inside that artery that's smaller than a  
4 pencil, and then --

5 Oh, once we have the wire down, we then advance  
6 this balloon over that wire. And when we are just  
7 across the blockage, watching again under the  
8 radiation of the fluoroscope, we blow up the balloon  
9 and we measure the pressure anywhere from just a  
10 couple all the way up to 20 atmospheres of pressure,  
11 and the balloon crimps open the artery at that point.

12 Now that's how big the artery is. I mean the  
13 picture makes it look much better. But that's the  
14 small space that we're working in.

15 Q. Doctor, are there risks to this procedure?

16 A. There are risks any time -- and that's why we  
17 take --

18 Even doing an angiogram, the diagnostic  
19 procedure itself has a one in 300 to one in 500  
20 chance of having a heart attack or stroke, about a  
21 one in 1,000 chance of dying from the procedure. And  
22 when we do an angioplasty there's about -- at the  
23 Minneapolis Heart Institute we're about 90 to 95  
24 percent successful with the initial balloon. In  
25 about one to two percent of cases, when we blow up

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1 the artery -- again, think we're working about an

2 arm's length away -- the artery can tear, and if we  
3 can't fix it, the patient needs to go to the  
4 operating room for an emergency bypass surgery.

5 So again these -- these are lifelong diseases  
6 that have taken many, many years to manifest  
7 themselves. None of these are cures. In the  
8 appropriately selected patients they are very, very  
9 good treatments, but they're not cures.

10 Q. Doctor, do you find that patients who have had  
11 an angioplastic procedure come back and the condition  
12 that they originally presented with has reoccurred?

13 A. Yes. And angioplasty is an injury, just like  
14 smoking is an injury, to the artery. But it pushes  
15 it open very, very wide. About 30 percent of people  
16 form a scar between two and six months after that  
17 initial injury where the artery comes back and  
18 narrows in, and they re-present with symptoms again.  
19 And then we either have to decide at that point  
20 whether we're going to stretch the artery again --  
21 there's only about a 50/50 chance of success -- or  
22 whether the patient at that time will need bypass  
23 surgery.

24 So again, the Achilles heel of angioplasty has  
25 been something called restenosis or rescarring of the

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1 artery back in.

2 Q. Doctor, what is the typical cost of a coronary  
3 angioplasty?

4 A. The typical cost of a -- of a coronary  
5 angioplasty is about 16,000 dollars.

6 Q. Doctor, then turning your attention to the stent  
7 procedure for a moment, if you would look in your  
8 book at Trial Exhibit 30025.

9 Does that depiction accurately depict a stent  
10 procedure?

11 A. Yes.

12 MS. NELSON: Your Honor, we would offer  
13 30025 for illustrative purposes only.

14 MR. MARTIN: No objection, Your Honor.

15 THE COURT: Court will receive 30025 for  
16 illustrative purposes.

17 BY MS. NELSON:

18 Q. And doctor, looking at 30002 and 30006, are  
19 those both examples of stents?

20 A. Two and four.

21 Q. 30002 and 30004.

22 A. Yes.

23 MS. NELSON: Your Honor, we would offer  
24 30002 and 30004 for illustrative purposes only.

25 MR. MARTIN: No objection, Your Honor.

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1 THE COURT: Court will receive 30002 and  
2 30004 for illustrative purposes.

3 BY MS. NELSON:

4 Q. Now Dr. Graham, looking at the procedure on the  
5 screen and utilizing the stents themselves, can you  
6 describe to the jury what a stent procedure involves.

7 A. Stent --  
8 Angioplasty is about 12 to 14 years old now.  
9 Stents have been the first real improvement over  
10 angioplasty in that when we place a stent, it -- it's  
11 actually like a little scaffold. It's deployed over  
12 a balloon, and the balloon is -- the stent is crimped  
13 on the balloon. The balloon, just as the angioplasty  
14 balloon that you're holding now, is delivered across  
15 the blockage, but what happens is it scaffolds the  
16 artery open. So when we finish an angioplasty, by  
17 the time the patient leaves the room, being it is  
18 living tissue, there is some elastic recoil, and then  
19 if you get that scarring restenosis, it closes down  
20 tight. In the stent when you leave the room, the  
21 artery remains scaffolded open by a piece of steel.  
22 And when that happens, even if it scars back in, we  
23 tend to still have an adequate blood flow down the  
24 artery.

25 The benefit of stents is that it has decreased  
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1 the restenosis rate, or that scarring back in, from  
2 about 30 percent down to about 10 percent.

3 I have a couple of stents here. Again, this is  
4 a -- a stent that is working to show you somewhat of  
5 the magnitude of the sizes that we work with. That's  
6 the stent that's expanded that would go in the  
7 coronary artery. This is a stent that's a little  
8 larger that is placed in the coronary artery. The  
9 company made a magnifying glass so you can actually  
10 see it. But it's a little piece of woven wire that  
11 goes in the artery, holds that artery open, and that  
12 has been a very major benefit for us in the  
13 catheterization laboratory.

14 Q. Now turning your attention to another procedure  
15 calls extraction atherectomy, would you look in your  
16 book to Exhibit 30024.

17 A. Yes.

18 Q. Is that an accurate portrayal of extraction  
19 atherectomy?

20 A. Yes.

21 MS. NELSON: Your Honor, we would offer  
22 30024 for illustrative purposes only.

23 MR. MARTIN: No objection, Your Honor.

24 THE COURT: Court will receive 30024 for  
25 illustrative purposes.

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1 BY MS. NELSON:

2 Q. Now Dr. Graham, can you describe this procedure  
3 for attempting to remove blockage.

4 A. Yes. The -- the atherectomy chamber has a  
5 cutting-head gear and a rotating blade that, as it  
6 goes across a blockage, actually then traps  
7 the debris here and we actually take it out of the  
8 body. One of the angiograms I'll show you, we  
9 actually used an atherectomy catheter such as this.

10 It's a big device. We can't use it on a bend  
11 over here, it has to be used on a straight. And we

12 need even a bigger tube than I showed you there to  
13 place it in the groin. So it -- it's a tool that we  
14 use, and -- and what is happening in the  
15 catheritization laboratory, we're kind of like a  
16 carpenter where we used to only have a hammer, and  
17 that's an angioplasty balloon, we now have stent,  
18 atherectomy devices, Roto-Rooters and things like  
19 that, so we have plyers and screwdrivers and the rest  
20 of the things too.

21 Q. Now this rotating cutting blade, is that like a  
22 knife that you're putting into the artery?

23 A. It's exactly like a knife.

24 Q. Okay. What does that procedure cost, doctor?

25 A. This procedure, because of the enhanced

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1 equipment, it actually uses a mechanical cutter,  
2 costs about 18,000 dollars.

3 Q. And are there risks to this procedure?

4 A. The biggest risk from this procedure, in about  
5 one percent of cases, that you would cut the wall of  
6 the artery, and then you would -- the patient would  
7 need to go to the operating room. Again, that's why  
8 we've learned not to do it on bends, we just do it on  
9 straightaways so that the cutter doesn't go out into  
10 the -- into the wall of the artery.

11 Q. Turning your attention now to coronary rotoblade  
12 procedure, Trial Exhibit 30027, is that an accurate  
13 portrayal of that procedure, doctor?

14 A. Two seven?

15 Q. There you go.

16 A. Yes.

17 MS. NELSON: We would offer, Your Honor,  
18 30027 for demonstrative purposes only.

19 MR. MARTIN: No objection, Your Honor.

20 THE COURT: Court will receive 30027 for  
21 illustrative purposes.

22 BY MS. NELSON:

23 Q. Can you explain what the coronary rotoblade  
24 procedure is, doctor.

25 A. This is the rotoblader that many patients think

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1 that they should have, but again when -- when the  
2 burr comes through here, it twirls a diamond bit  
3 here. Each of these bits cost about seven to eight  
4 hundred dollars. Goes across the lesion, and it  
5 works well in people who have heavy calcified  
6 lesions, where the lesions are like rocks, "lesions"  
7 meaning blockages.

8 Again the problem with the rotoblader is when we  
9 chop some of this up, some of it goes downstream. It  
10 can cause muscle damage downstream in the heart and  
11 you need big arteries to do this in. Because of  
12 that, again, this has been kind of a niche player.  
13 Again, we select the right tool that's appropriate  
14 for the right job.

15 Q. And doctor, what is the typical cost of this  
16 procedure?

17 A. Also about 18,000 dollars.

18 Q. Turning your attention then to coronary bypass  
19 surgery, if you would look at the model 30016, could  
20 you explain to the jury what coronary bypass surgery  
21 is.

22 MS. NELSON: Your Honor, we offer 30016 for  
23 demonstrative purposes only.

24 MR. MARTIN: No objection, Your Honor.

25 THE COURT: Court will receive 30016 for  
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1 illustrative purposes.

2 A. If there are blockages in many vessels, and  
3 there -- we will sometimes, and unfortunately too  
4 often, do coronary artery bypass surgery. And if  
5 there's a blockage in the right coronary, the left  
6 anterior descending and the circumflex, where there  
7 is just too many to do balloons on and the patient  
8 has on going symptoms or is at risk, we think, of  
9 dying from his or her disease, the surgeon will then  
10 open the chest and then place -- and stop the heart,  
11 the patient will be placed on an oxygenated heart/  
12 lung machine to bypass the heart to give the surgeon  
13 time to work on that, and then we'll take a vein from  
14 the leg and bypass around the most serious blockages.  
15 We'll oftentimes use what we have call the left  
16 internal mammary artery to bypass the LAD. It's not  
17 depicted on this model. But a bypass is a detour  
18 around the blockages.

19 We know in post-bypass stage that if patients do  
20 not address their causative agents and they continue  
21 to smoke, if they have high cholesterol, that these  
22 grafts, nearly 60 percent of them will be gone by 10  
23 years after they -- after the surgery. And we take a  
24 vein, which is a very low pressure system, and put it  
25 under that arterial pressures, remember that left

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1 ventricle is pumping very hard and it pounds that,  
2 and if there are injuries to the vein, the vein can  
3 then close and the patient would have a heart attack.

4 Q. Dr. Graham, what is the most frequent major  
5 surgery in the United States?

6 A. Unfortunately, coronary bypass surgery has  
7 become the most common procedure, major surgical  
8 procedure in the United States. Over 400,000  
9 performed last year.

10 Q. And what are the risks of this procedure?

11 A. If you have a good pump going in, your surgical  
12 risks are usually one to two percent. Depending on  
13 how well the heart pumps going in, your risks go up  
14 as high as 20 to 30 percent mortality.

15 If somebody comes in on a helicopter, we do an  
16 emergency angiogram, we see that many vessels are  
17 blocked, and their ejection fraction is 20 percent,  
18 normal being 60 percent, meaning how much blood the  
19 heart ejects with each squeeze, and then we rush them  
20 to the operating room in order to try and save their  
21 lives, you know, we may lose two or three out of 10

22 of those patients. We save seven or eight of them,  
23 but we still lose patients. And we try to do  
24 everything we can, but, you know, this isn't  
25 tiddlediwnks. We -- patients, even when we do  
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1 everything as best we can, the crime is those  
2 patients die right in front of our face, and we have  
3 to deal with them.  
4 Q. Doctor, what are the major risk factors for a  
5 poor outcome of coronary bypass surgery?  
6 A. In the short term, as I just mentioned, the  
7 worst outcome is left main coronary artery disease,  
8 the main artery going into the heart and the ejection  
9 fraction. Long term, the two things that have been  
10 closely related to lack of survival of these bypass  
11 grafts or closing of them, which are now the  
12 patient's lifeline, are smoking and high cholesterol.  
13 Q. What happens when a patient comes to you, a  
14 smoker comes to you with COPD and requires coronary  
15 bypass surgery?  
16 A. Again, as we talked about the risk of -- of  
17 bypass surgery and as we talked about the causative  
18 agents of why smoking was bad, if you have somebody  
19 now who is in the midst of an unstable situation and  
20 they have lung disease, like Dr. Davies outlined,  
21 even if it has not been a clinical problem to their  
22 life, they have an organ system that is on the blink,  
23 they need lung reserve like they've never had before.  
24 Patients who have scarred their lungs, even if it  
25 hasn't been a problem to that time, are now -- their

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1 risk of bypass surgery goes up two to three times  
2 because of their lungs. They tend not to be  
3 extubated or get the tube out after they've had  
4 bypass as quickly. The longer that they are kept on  
5 the breathing machine, the more chance they have of  
6 pneumonia. There are just many, many risk factors  
7 that go along with smoking. And what may be  
8 subclinical, which means it has not presented as to  
9 the doctor's saying I have COPD, but with the  
10 stresses and the need for the extra reserve of their  
11 lungs, it isn't there.  
12 Q. Doctor, what is a typical cost of coronary  
13 bypass surgery?  
14 A. Typical cost in the Twin Cities area of bypass  
15 surgery is approximately 30,000 dollars.  
16 Q. Thank you, doctor. You can return to the  
17 witness stand.  
18 Now you have a patient who has recovered from  
19 one of these life-saving procedures, the angioplasty  
20 or the rotoblade or coronary bypass, and has  
21 stabilized. At this point what interventions are  
22 available?  
23 A. Once a patient's life has been saved and --  
24 and -- and I would stress that, you know, it's --  
25 it's often a life and death situation, the long-term

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1 prognosis of that patient is to keep their blockages  
2 that they have now in their coronary arteries from  
3 progressing. Somebody who has had a heart attack and  
4 was in the lucky 70 percent to make it to the  
5 hospital the first time, if they have a second heart  
6 attack, their out-of-hospital mortality rises not  
7 from 30 percent but to 50 percent. Okay. So we do  
8 everything we can to keep that patient out of a  
9 crisis, which is both medically expensive and life  
10 threatening to the patient.

11 So if the patient smokes, we do everything in  
12 our power to try and get them to quit smoking. If a  
13 patient has high cholesterol, we educate them in  
14 diet, in exercise to increase HCL, the good  
15 cholesterol, and they may not pharmacologic or drug  
16 treatment to lower their cholesterol. If a patient  
17 has diabetes, we very aggressively treat their  
18 diabetes to normalize the blood glucose or the blood  
19 sugar. If there are certain drugs, again, that have  
20 been shown if a patient has heart disease to keep  
21 them alive, the patient will be on aspirin the rest  
22 of their lives, a class of drugs call beta blockers  
23 which has been shown to decrease the incidence of  
24 death after a heart attack, a classic drug called an  
25 ace inhibitor, which makes -- decreases the strain on

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1 the heart and makes it easier for it to push forward,  
2 especially in those patients who are prone to what we  
3 call congestive heart failure. So the patient now  
4 becomes a high-maintenance patient.

5 What we want to do for a number of reasons is to  
6 keep that patient out of, again, another crisis of  
7 medicine.

8 Q. Are those long-term drug therapies costly?

9 A. They can be very costly.

10 Q. Now a patient who has bypass surgery, does that  
11 patient need cardiac rehabilitation?

12 A. The standard of care in Minnesota -- and we are  
13 very strong believers in working with our primary  
14 care counterparts throughout the state -- is to have  
15 an aggressive cardiac rehab program. We are very  
16 efficient in getting patients in and out of the  
17 hospital, so efficient that sometimes they -- they  
18 don't remember what we tell them because they are  
19 still under the effects of some of our medications  
20 when they -- by the time they leave, but then working  
21 through cardiac rehab, the messages of stopping  
22 smoking, of treating cholesterol, of exercise, are  
23 reinforced in a structured fashion that has been  
24 become standardized and is very beneficial to the  
25 patient.

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1 Q. And is cardiac rehabilitation costly, doctor?

2 A. Yes. Most patients will undergo anywhere from

3 18 to 36 sessions.

4 Q. Now doctor, once the patient has suffered a  
5 heart attack, and as a consequent death of a portion  
6 of the heart muscle that you showed us before, is  
7 that patient at increased risk for other diseases?

8 A. The heart muscle once it dies -- in its living  
9 state it conducted electricity in a very regular way,  
10 and that's how the heart beats. There's an  
11 electrical wave that crosses the heart that causes  
12 the muscle to twitch, just like a nerve in our hand,  
13 it really is an electrical wave that causes the  
14 muscles to twitch.

15 When the muscle dies, the conduction through  
16 that becomes irregular, and if the beat happens at  
17 the right time, the patient's heart can start beating  
18 chaotically. If the pumping chamber of the heart  
19 does not beat, the blood pressure falls to zero and  
20 within six seconds the patient would pass out. So  
21 there is a considerable number of patients who have  
22 had heart attacks who then go out and die suddenly,  
23 what's called sudden cardiac death, many of you may  
24 know people who died in their sleep, it's not usually  
25 of a heart attack that they die, it's usually of an

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1 arrhythmia, which means a chaotic beating of the heart  
2 muscle so that it does not pump blood efficiently.

3 Q. And are there medical devices available today to  
4 lessen the risk of patients suffering from these  
5 misfired electrical impulses?

6 A. We know now that there -- if -- if this  
7 life-threatening arrhythmia is to happen, that there  
8 is no medication that can really save the patient, so  
9 because of that, in patients who we risk-stratify,  
10 that we think are the highest risk of that, patients  
11 again who have poor pumps, ejection fractions less  
12 than 40 percent, 60 percent being normal, we will  
13 then sometimes undergo what we call  
14 electrophysiologic testing. If we create what we  
15 believe is a life-threatening arrhythmia in a  
16 controlled circumstance, again in a somewhat of a  
17 cardiac catheterization laboratory, we will then  
18 implant what we call a cardiac defibrillator that has  
19 leads into the heart that will shock the heart with  
20 electricity if the patient has a life-threatening  
21 chaotic rhythm. This is what's been shown to be the  
22 only effective thing if the patient has a lethal  
23 arrhythmia.

24 Q. And do you also have available to you  
25 pacemakers?

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1 A. In certain patients, after a heart attack --  
2 again the conduction system of the heart is a very  
3 regular, as we think of just electrical circuits  
4 going from the top to the bottom of the heart. If  
5 the conduction system or if one of those wires that  
6 is going through the muscle is damaged after a heart  
7 attack, the patients may not conduct electricity from

8 the top to the bottom of the heart. We have then  
9 available electronic pacemakers threaded in to the  
10 right side of the heart that then causes the heart to  
11 beat in a regular fashion and keep a kind of a safety  
12 net so the heart doesn't go too slow. Very fortunate  
13 in Minnesota to have the three largest pacemaker  
14 companies in the world located here in Minnesota.

15 Q. Dr. Graham, have you brought an example of a  
16 real pacemaker and a real defibrillator to the court  
17 today?

18 A. I have.

19 Q. Could you step down a moment, with the court's  
20 permission.

21 MS. NELSON: Your Honor, we would offer  
22 30000 and 30001 for illustrative purposes only.

23 MR. MARTIN: No objection, Your Honor.

24 THE COURT: Court will receive 30000, 30001  
25 for illustrative purposes.

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1 BY MS. NELSON:

2 Q. Dr. Graham, would you describe both the  
3 pacemaker and the defibrillator to the jury.

4 A. The pacemaker is a marvel of modern electronics.  
5 The -- this is made by Medtronic, a Minnesota  
6 company. That the leads then are -- this is placed  
7 below the skin. We place a needle in a vein and then  
8 we thread these leads down into the heart, and this  
9 then resides under the skin here. This can be  
10 programmed to go faster, slower, to speed up when you  
11 walk, things like that. And the electronic engineers  
12 are marvelous people.

13 The leads -- there's two leads. The pacemaker  
14 itself costs about 10,000 dollars. Each lead --  
15 there should be two leads on this -- about 600  
16 dollars.

17 Q. And the defibrillator.

18 A. This is what some people would call a shock box.  
19 It can either deliver a fast burst of electricity or  
20 a committed shock to the heart. This is hooked to a  
21 series of leads also that are threaded through the  
22 venous side into the right side of the heart, and  
23 then it's placed under the skin here. It also can be  
24 interrogated to see what it's done over the past  
25 time. This is made by CPI, again another Minnesota

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1 company. This device costs about 25,000 dollars.  
2 And with that, leads -- there's four leads in  
3 here -- they cost about 600 dollars each.

4 Q. Take a look at 30020 again, please. Doctor,  
5 what is --

6 Is congestive heart failure another risk posed  
7 to a patient who has already suffered a heart attack?

8 A. As we discussed previously, after somebody has a  
9 heart attack there's a critical amount of muscle mass  
10 that we all need to push blood forward. If we lose  
11 that muscle mass, the heart becomes an inefficient  
12 pump, and then we have to give medications and all in

13 order to try to compensate for the heart not pumping  
14 well.

15 Q. What is the most common reason for admission of  
16 an adult to an American hospital?

17 A. The most common reason for admission of adult  
18 population in the United States is congestive heart  
19 failure.

20 Q. Doctor, can the treatment of congestive heart  
21 failure be complicated by COPD?

22 A. When people are short of breath, again as I  
23 mentioned before, it's often a question: Is it the  
24 heart or is it the lung? If somebody has baseline  
25 mild shortness of breath from mild lung disease,

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1 although they had never presented to the doctor for  
2 it, then they come in and have had also the question  
3 of heart failure, it becomes oftentimes a diagnostic  
4 dilemma. Is it -- is it COPD causing the shortness  
5 of breath, or is it heart failure? And sometimes in  
6 those patients we place what's called a Swan-Ganz  
7 catheter that measures the pressures on the right and  
8 indirectly the left side of the heart, so that we  
9 know whether to give those patients more or less  
10 fluid and helps us make the diagnostic decision  
11 whether this is heart failure or whether the lungs  
12 are the problem.

13 Q. Now doctor, you explained to the jury at the  
14 beginning that the Heart Institute also performs  
15 heart transplants.

16 A. Yes.

17 Q. Could you describe to the jury the process of  
18 cardiac transplantation.

19 A. Cardiac transplantation is something we reserve  
20 to patients -- for patients who have no other  
21 options. Transplantation, taking immunosuppressive  
22 drugs, waiting on a list, waiting for a heart  
23 transplant, is no bowl of cherries. And there's  
24 always a risk that the body will reject the heart and  
25 the immunosuppressive drugs need to be titrated.

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1 We do approximately 25 transplants a year at the  
2 Minneapolis Heart Institute. Half of the patients  
3 who have a heart transplant have had heart attacks  
4 previously; the others have had various other  
5 sicknesses of the heart where their heart no longer  
6 functions.

7 With that, you know, the expense of waiting for  
8 a heart transplant sometimes can be the -- the  
9 biggest -- as big an expense as the actual operation  
10 itself. We just yesterday transplanted a man who was  
11 waiting 75 days in our coronary care unit for a heart  
12 as a status one heart transplant. And it's  
13 maintaining that very delicate balance of a -- of a  
14 compromised heart. We have another man who is  
15 approaching 60 days and is there now. And the  
16 problem is the donors, we don't have the donors.

17 Q. And what is the cost of the typical heart

18 transplant?

19 A. The yearly cost of a heart transplant would  
20 approach, the first year, over a hundred thousand  
21 dollars.

22 Q. Doctor, I want to turn your attention back to  
23 30018. We've been addressing blockages in the  
24 coronary arteries of the heart. Are there other ways  
25 in which arterial blockages occur in the body?

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1 A. Much the same as the risk factors and causative  
2 agents that led to blockages in the heart and what we  
3 think is very, very important -- because if the pump  
4 doesn't work, nothing works -- atherosclerotic or  
5 blockages in the carotid arteries leading to the  
6 brain as well as -- which would lead to stroke, the  
7 other common cause of strokes is after somebody has  
8 had a heart attack, if they form blood clots in the  
9 heart from part of the heart not working well, pieces  
10 can break off and go north into the head and cause a  
11 stroke.

12 Remember, the brain is the most sensitive organ  
13 in the body as far as deprivation of oxygen. Six  
14 seconds is all it can stand. So stroke is a -- is a  
15 debilitating, terrible disease, and especially in our  
16 older population, most of our patients are probably  
17 more afraid of stroke than they are of dying.

18 Q. And what are the treatments available for  
19 someone who has had that sort of clot and the  
20 consequent stroke?

21 A. Unfortunately, patients oftentimes, when they  
22 presented with stroke, they -- they are far down --  
23 too far out to -- to give much therapy. We have  
24 begun giving some patients the same type of  
25 clot-dissolving medicine for certain types of stroke,

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1 but they need to be rushed to a CAT scanner first to  
2 make sure that they have had no bleeding in their  
3 head, because if you gave a clot-dissolving medicine  
4 and the patient had that type of stroke that was from  
5 bleeding, you would make the bleeding worse. And  
6 then once that happens, the -- if the patient has not  
7 been salvaged from that or has had an incomplete  
8 stroke, sometimes they will have a carotid  
9 endarterectomy, which is a surgery that essentially  
10 peels the plaque out of the carotid artery, with  
11 about a 10 percent incidence of stroke just from the  
12 procedure itself.

13 The worst thing about stroke is most  
14 patients -- the right side of the brain runs the left  
15 side of the body and vice versa. Oftentimes we have  
16 a very debilitated patient who, as I said, especially  
17 in our older patients who are faced with stroke, are  
18 more afraid of that debilitation than actually dying.

19 Q. And can that debilitation, doctor, often lead to  
20 nursing home care?

21 A. Again, in a -- in a patient who half of his or  
22 her body does not work oftentimes needs heavy levels

23 of care. There is usually an acute hospitalization,  
24 a transitional-care unit, which is a step down within  
25 the hospital or a like unit, and then oftentimes

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1 chronic nursing home care of some sort.

2 Q. And then looking at the lower portion of the  
3 body, the aorta and down, do you find that there's  
4 blockages in those peripheral vascular locations?

5 A. Again, peripheral vascular disease --

6 "peripheral" meaning downstream, especially in the  
7 legs -- happens to two groups of people, smokers and  
8 diabetics. So people whose blood glucoses are high,  
9 blood sugars are high, and smokers, are at risk for  
10 developing peripheral vascular disease. While it  
11 again is a terribly debilitating disease and tends to  
12 be a more diffuse disease all up and down the artery,  
13 and so by-passing around those or doing a balloon on  
14 those oftentimes is very difficult. Plus the  
15 arteries are very small as they go down, especially  
16 below the knee. Can then sometimes lead to  
17 amputations, multiple surgeries in order -- if the  
18 blood supply is there, that the wounds don't heal  
19 well. So it's a very, very morbid thing to have.

20 The other side of the coin is people usually  
21 don't die of peripheral vascular disease. But if  
22 somebody had symptomatic blockages in their legs, and  
23 if they walk in and tell me that they have pains in  
24 their legs when they walk a block or two, so it's  
25 fairly symptomatic, those patients statistically have

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1 a 75 percent 10-year cardiac mortality. Because I  
2 know if there's blockages here, there's almost  
3 certainly blockages here.

4 MS. NELSON: Your Honor, this point would  
5 be a good time to break for the evening.

6 THE COURT: All right. We'll recess,  
7 reconvene tomorrow morning at 9:30.

8 THE CLERK: Court stands adjourned until  
9 tomorrow at 9:30.

10 (Court recessed.)  
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